

**POLIOMYELITIS
AND
POLIOENCEPHALITIS**

POLIOMYELITIS AND POLIOENCEPHALITIS

by

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**This book is dedicated to
MY FAMILY**



PREFACE

POLIOMYELITIS and polioencephalitis are endemic throughout the world, but periodically they become epidemic and unlike the other infectious diseases they appear to be increasing in frequency. The first major epidemic of this acute infective disease occurred in Great Britain in 1947, and it was followed by a second in 1949. From previous experiences in other countries which have been ravaged by this disease, it is certain that these two epidemics will be followed by others.

In 1947 the majority of the medical profession in this country was quite unfamiliar with the diagnosis and treatment of any large numbers of patients suffering from poliomyelitis, and the advisability of establishing special centres throughout the country had to be considered. As the number of admissions of service patients had greatly diminished since the end of the second world war, it was suggested that the orthopaedic and rehabilitation centre at Pinderfields General Hospital, Wakefield, should be placed immediately at the disposal of the victims of this dread disease, and a poliomyelitis unit was established.

The popular term "infantile paralysis" should, in my opinion, be strictly avoided for the following reasons:

- (1) The disease is by no means confined to infants: the tendency during recent epidemics has been for a much larger proportion of adolescents and adults to be the victims
- (2) This infection is only one of the numerous causes of paralysis found in infants
- (3) Only a small proportion of the affected patients develop paralysis

Indeed, judging from experience in the years 1947 and 1949, of every 100 suspected patients, 5 or 6 are likely to die; 9 or 10 to be severely paralysed; 17 or 18 to have some degree of residual paralysis, 35 or 40 to suffer either no ill-effects or to be left with a slight degree of paralysis, whilst in the

cerebrospinal fluid cell count with a preponderance of polymorphonuclears in the first few days followed later by a lymphocytosis and a diminished polymorphonuclear count by the end of the second week.

Against a diagnosis of poliomyelitis we noted:

- (1) Persistent absence of changes in the cerebrospinal fluid.
- (2) Persistent absence of pleocytosis or a pleocytosis over 1,000/cm.
- (3) Values of albumen over 200 mgm. %.
- (4) Decreased quantity of sugar, especially a progressive decrease of sugar during the illness.

BLOOD GROUPS

We failed to find that any particular blood group was unduly susceptible to infection by poliomyelitis.

MUSCLE

In our series it was soon obvious that the Medical Research Council's grading of muscle power was simple, required no apparatus and was remarkably accurate.

For the examination of muscle power in any one muscle or muscle group, we advise that it should not take more than two to three minutes, and that it should not be repeated more than twice in twenty-four hours.

In young children we encountered considerable difficulty in muscle testing cases in which muscular contractions were already present.

Jerky movements of the limbs were noted in 22% of our cases and twitching of individual muscles in 12%.

Pain in the limbs occurred in 18% of cases.

Pain and tenderness on stretching a muscle group was not a common finding and is usually observed after the paralysis is well established.

Muscle tenderness lasted in the majority of our cases for periods of from one day to one week whilst the minority lasted up to five weeks.

Muscle tenderness was particularly noted in sixteen cases

Thus:

- 5 cases had slight tenderness with severe subsequent paralysis
- 6 cases had fairly severe tenderness with no subsequent paralysis.
- 3 cases had fairly severe tenderness with slight subsequent paralysis
- 2 cases had slight tenderness with slight subsequent paralysis

In our opinion, there are two types of muscle spasm

- (1) Early spasmodic which occurs in the acute phase and lasts for a few days. In other words it is fairly transitory
- (2) This type persists for more than a month and is very troublesome. It appears to be a reflex contracture due to a pain stimulus, and is frequently accompanied by stiff joints and contractures.

The various theories of muscle spasm are discussed

There appears to be no relationship between the degree of paralysis and the frequency and intensity of muscle spasm

There is no definite proof that the relief of muscle spasm prevents paralysis or alters the mortality in the acute stage.

Spasm temporarily impairs muscle function and destroys the normal reciprocal relation between agonists and antagonists

Marked rigidity was present in 22% of cases, Kernig's sign in 20% of cases, head retraction in 18% and stiff back in 62% of cases.

Muscle spasm was aggravated by massage, premature weight bearing and exposure to cold

PARESIS AND PARALYSIS

Fifty per cent of our cases were of sudden onset and 50% were gradual.

Muscular weakness in poliomyelitis has been attributed to several causes some of which are enumerated in the text.

The muscles appear to be involved in proportion to their

house surgeons and house physicians, physiotherapists and hospital nursing staff, but it is earnestly hoped that many other readers will find some sections of great use to them.

In the matter of illustrations and photomicrographs, I am greatly indebted to Mr. N. L. Capener, F.R.C.S., Dr. G. H. Jennings, M.D., F.R.C.P., and Dr. W. Howlett Kelleher, M.D., D.P.H., and also to the firms of Siebe, Gorman & Co. Ltd., Stanley Cox Ltd., and Spencer (Banbury) Ltd.

I wish to thank my wife who has typewritten the manuscript and whose encouragement and help has greatly lightened my task.

Finally it is a pleasure to express my thanks to my publishers, Messrs H. K. Lewis & Co. Ltd., for their constant courtesy and consideration.

R. A. RUSSELL TAYLOR

Leeds,
December, 1954

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CHAPTER I

EPIDEMIOLOGY

FIRST of all it must be stated that unlike other infectious diseases poliomyelitis is increasing both in frequency and severity

The first authentic evidence of poliomyelitis ■ thought to be that manifested in a five-thousand-year-old Egyptian skeleton in which one leg was affected. It was not, however, until the beginning of the nineteenth century that this disease became recognized as a clinical entity.

Although small epidemics were reported upon before 1868, the first large epidemic occurred in Scandinavia in that year. Other large epidemics were later noted in the United States of America, North-Western Europe, Iceland and Australia. Epidemics were thought to occur only in temperate zones, but since the end of the nineteenth century, there has been a steady increase in the number and extent of these epidemics throughout the whole world.

During the second world war, observers found that poliomyelitis was a common infection in the tropics, and an important point noted was that the white troops were much more severely affected than the native troops. McAlpine (1945) also made the observation that British officers were affected five times more frequently than other ranks. It has been suggested that whereas officers, even ■ the field, frequently ate their meals in a mess where crockery was communal, other ranks ate from their individual mess tins.

It has been shown that in countries where there ■ a mixed population, Europeans were less immune than the indigenous natives, but there was an equal incidence in the case of very young children. I would suggest that this may be explained by the fact that the natives were exposed much earlier and more frequently to the disease prevalent in their area.

Also it was observed that American troops based in the Philippine Islands were affected ■n times more frequently than home based troops, whilst the native population

survive more easily outside the body. During the summer months when the schools are closed and people spend a great deal of their leisure time out of doors, the spread by droplet infection is considered to be a less likely cause. Most investigations tend to show that although poliomyelitis occurs in an epidemic form at all seasons of the year, the number of paralytic cases is much higher during the summer months. It is interesting to note that the seasonal epidemic graphs of poliomyelitis, gastro-enteritis and typhoid fever are very

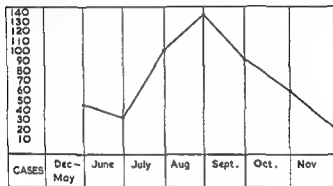


FIG 1

similar. Bowerman (1945) made the important observation that the outbreaks of poliomyelitis seemed to occur after long periods of dryness and increased temperature. Recently it has been suggested that the loss of chlorides from sweating may explain the seasonal physiological changes in the host resulting in a lowered resistance, but no definite conclusions on this point have so far been reached.

Poliomyelitis diminishes markedly in frequency in the early winter months in contradistinction to the other respiratory diseases, but epidemics can occur during the winter months, as reported upon by Ward and Sabin in 1944. Other examples are the severe epidemics in Iceland, the Malta epidemic in 1942-43 and the two outbreaks amongst the Canadian Eskimos in the severe winter of 1948-49.

The rise and fall of the epidemic curve of poliomyelitis in

remained relatively immune. The assumption appears to be that the Americans had reached adult age without acquiring immunity, and that they had therefore contracted the disease at a much higher rate.

It is therefore reasonable to conclude that epidemics will occur in any country where large numbers of children fail to acquire immunity to the virus of poliomyelitis during the first five years of life.

As pointed out by Paul (1947), poliomyelitis is endemic in Japan, but it was not until 1948 that an epidemic occurred there. The recent epidemics in other countries have been attributed to the introduction of the invasive strains of the virus by returning troops from Malta, France, Germany, etc.

Poliomyelitis attacks the vast majority of people in these endemic areas at some period of their lives, but it is usually so mild that they are almost free from symptoms, and according to Casey (1948), only about 2% of all cases develop residual paralysis. During epidemics, on the other hand, American observers estimate that 30% to 60% of all cases have permanent evidence of paralysis.

The incidence and severity of the disease appears to be greater with increasing distance from the Equator, whilst the extent of the disease and the symptoms differ from individual to individual, and in different epidemics.

The seasonal incidence was first commented upon by Sinkler (1875), and in 1878 Barlow noted that 27 out of 53 cases collected over a period of seven years occurred during July and August.

In the United States of America, the epidemic spreads northwards reaching its peak in September and commencing to die out in October, whilst in the southern hemisphere the peak months are January to April.

In this country, the highest incidence of poliomyelitis has occurred during July, August and September. The graph opposite shows the month of onset of the disease in my series of cases.

At this time of the year, flies are numerous and contaminated food and water are more liable to cause an epidemic, because during warm weather the organisms will

only a very small percentage of infected cases develop paralysis, and many temporary carriers do not contract the disease owing to their lack of susceptibility. The presence of the virus in washed portions of the tongue, posterior pharyngeal wall, bronchi, oesophagus, stomach, duodenum and various levels of the large and small intestines has been demonstrated. Certain observers are strongly of the opinion that the pharynx is the portal of entry, but whether the infection occurs primarily by inhalation or by ingestion remains a matter of conjecture.

Howe and Bodian (1947) succeeded in isolating the virus from the throats of symptomless patients and a year later showed that these children had developed antibodies of the virus which had been isolated from them.

The active virus is present in the nasopharynx for approximately ten days after the onset of clinical symptoms, although Casey (1942) considered that the effective reservoir of the virus in a patient was within three days before and three days after the onset of the first prodromal symptoms. Other observers have given these times as six days before and possibly a slightly longer period afterwards. The following table shows the number of days between the onset of the illness and the first appearance of paralysis in various age groups in our series of cases.

TABLE I

Days	Age groups		
	0-5 years	5-15 years	15+
1	11	5	8
2	6	6	13
3	9	5	9
4	6	2	3
5	6	4	5
6	4	0	2
7	3	1	1
8-21	6	2	6

The virus may also be isolated from the nasal washings in abortive cases and in convalescents

quite characteristic. Within a given area, the incidence rises progressively for six to eight weeks, and then slowly declines. The exceptions to this have been in the Malta epidemic of 1942-43, and also in Singapore and Mauritius where a sudden rise and an equally sudden fall in the number of cases was experienced.

When the disease is endemic, the sources of infection may not be discovered, whilst in an epidemic the infective agent is so widely distributed that the innumerable temporary carriers cannot be detected. In the early stages of the outbreak, direct or indirect contact between many of the cases may be demonstrated, but in the later stages this is impossible. The frequency of the infection amongst contacts appears to be proportional to the closeness of their contact with a proved case.

Bryant (1910) regarded epidemic poliomyelitis as a contagious and infective disease, the causative organism of which entered the system by means of the nasopharynx. In the same year, Flexner and Lewis infected monkeys by the nasopharyngeal route and recovered the virus from the cerebrospinal fluid on the third day. Batten (1913) expressed the opinion that the virus was carried in the mucous membrane of the mouth, nose and pharynx, not only in those suffering from the disease but also in the contacts.

Kling and Levaditi (1913) and McNalty (1938) have shown that infection in the human invariably spreads by case to case contact, especially when people are congregated in such places as buses, railway carriages, schools, theatres and cinemas.

Casey *et al* (1945) have shown that minor illnesses or mild abortive cases are four to six times more common among associates of a known case than among controls. It should also be noted that the virus may be isolated from the throat or stools of apparently healthy contacts, but a follow-up study by certain observers has failed to reveal any persistent carriers.

Infectivity is greatest during the latter part of the incubation period and in the febrile preparalytic stage in patients or carriers. This is obscured by the fact that in any epidemic

described by Anderson (1945), where in 43% of bulbar and bulbo-spinal cases tonsillectomy had been performed within thirty days preceding the onset, but Cunning (1947) in the series he investigated, found the incidence of similar cases to be only 2.5%. Previously, in 1946, Cunning had found that there was no causal relationship between poliomyelitis and tonsillectomy. When poliomyelitis has followed tonsillectomy, adenoidectomy or an associated tonsillitis, it is possible that the trauma or bacterial infection may open up peripheral neural pathways for the invasion of the virus. The virus passes up the exposed afferent fibres of the sensory cranial nerve or to the posterior spinal root ganglion, and from there to the neighbouring motor neurones. Anderson *et al.* (1950) stated that the risk of developing poliomyelitis was at least three times as great as in the normal population.

times as great. This was confirmed by Seigel (1951) who stated that the most desirable months for elective operation seem to be the winter months which are farthest removed from the poliomyelitis season. However, the growing weight of evidence appears to be against the viewpoint that tonsillectomy predisposes very seriously to the development of poliomyelitis, but a number of American authors still hold the view that there is a higher percentage of cases with bulbar involvement after tonsillectomy.

Flexner and Lewis (1910) put on record that they found the virus in the mesenteric glands in a human case, and other observers have also found it in the abdominal viscera. It was not until 1936 and again in 1945 that Toomey expressed the now generally accepted opinion that the whole clinical picture could be accounted for by the entry of the virus through the alimentary tract and its spread by the sympathetic and parasympathetic fibres to the central nervous system and peripheral nerves. As previously mentioned, the virus is present in the pharyngeal secretions only during the acute stage of the disease, but it can also be demonstrated in the stools of patients, contacts, convalescent subjects and healthy carriers some of whom later may develop the disease.

Fairbrother (1935) expressed the opinion that the nasopharynx is the portal of entry and that the spread to the anterior horn cells occurs through the brain tissue by way of the axis cylinders. As the result of further experiments on monkeys, Toomey (1936) suggested that the virus could travel along the chorda tympani to the superior salivary nucleus and from there to the seventh cranial nerve in the medulla. Robertson (1940) after a study of the olfactory bulb from eleven fatal cases of poliomyelitis, stated that his investigations offered no definite evidence for or against the invasion of the nervous system by the olfactory path. Faber and Silverberg (1946) found that the Gasserian ganglion as well as the secondary centre of the trigeminal pathways were moderately severely involved in the majority of the cases they reviewed. The virus may also ascend from areas supplied by the fifth cranial nerve (afferent root) and other cranial nerves, as is seen in cases with rapidly fatal bulbar involvement with little or no paralysis of the limbs.

All the accumulated evidence seems to suggest that the virus may reach the central nervous system at an early stage when the bulbar palsy is greatest, by travelling along the glossopharyngeal nerves from the pharyngeal and tonsillar regions. Pathological signs may also be present which suggest a portal of entry through the visceral afferent system of the glossopharyngeal and vagus nerves, but almost any nerve tissue with peripheral nerve endings in the mucous membrane may convey the virus in a central direction to the regional ganglia.

Certain arguments have, however, been brought against the nasopharyngeal route being that of spread to the central nervous system. In the first place, patients dying of poliomyelitis seldom show any demonstrable lesions in the olfactory bulbs. Secondly, there is a definite increased incidence of polioencephalitis in patients who appear to contract the disease shortly after tonsillectomy. Aycock and Luther (1929) and Brown (1932) were the first to draw attention to this relationship, and their observations were confirmed by Fischer and Stillerman (1937 and 1941), and by Toomey and Krill (1942). An epidemic in Utah was

Several of our cases gave a history of diarrhoea, nausea and vomiting which they attributed to various articles of diet, e.g. pies, fish, meat paste, fresh fruit, etc. Further investigations will probably show that pharyngeal secretions prove to be the source of most contact infection, and faeces the chief source of virus in non-contact infection, sub-clinical immunization or perpetuation of epidemics.

The poliomyelitis virus is comparatively susceptible to the acidity of the normal gastric juice, but as this varies from individual to individual, the possibility of the virus reaching the duodenum in an active form must vary with the person. The virus may be so attenuated that it is incapable of penetrating the bowel wall and this may account for some variations in individual susceptibility. Ashworth (1948) proved that there is no evidence that the gastric acidity of the population with poliomyelitis has a different distribution from that of a normal population.

It has been suggested that some features of the disease are more readily explained if it is assumed that the virus spreads in the cerebrospinal fluid and that it may reach the central nervous system by the lymphatic channels. In the body as in the community, there may be several routes by which the virus spreads.

Summarizing the modes of spread, it is found that the principal recognized portals of entry are

- (1) From the nose, mouth and pharynx by the afferent fibres of the fifth, ninth and tenth cranial nerves
- (2) From the trachea and bronchi by the afferent fibres of the ninth and tenth cranial nerves, and the sympathetic ganglia
- (3) From the alimentary tract by the sympathetic nervous system to the coeliac ganglion

The virus is known to retain its activity outside the body up to ninety days in the cold or in water, and even two hundred days in faeces. Although often found in raw sewage during epidemics, it has not so far been detected before the outbreaks. At times, the activity of raw sewage has been so high as to suggest that at least 6% of the population were

In 1939 Lépine showed by inoculation into monkeys that the virus was present for 123 days in the stools of an apparently healthy child whose father was a victim of the disease, and that the blood serum of the child had a neutralizing action on the virus. McClure and Langmuir (1942) found the virus in the faeces of 4 out of 5 patients with definite clinical signs and symptoms of poliomyelitis, and in 20 out of 27 who had had intimate contact with these patients. The intestinal tract might be considered as a focus of virus proliferation, but it has been shown that the virus has been eliminated in diminishing quantities in each successive stool. In the Middle East in 1944, Paul attempted to isolate the virus from the faeces of British and American troops suffering from poliomyelitis and his results indicated that the amount of the virus present was greater in the more severe cases, and that the chance for positive results was greater in the early stages of the attack. Horstmann *et al.* (1944) detected the virus in the stools of patients as follows: 70% of patients in the first two weeks, 50% at the end of the fourth week, 27% at the end of the sixth week, and 12% at the end of eight weeks after the onset of the disease. The virus has also been found in the faeces two to three weeks before the onset of the paralysis and as late as seventeen and a half weeks after the initial symptoms.

It is therefore evident that there are two potential sources of infection, namely the nasopharynx and the gastro-intestinal tract. It has been stated that the virus is found in the stools in 70% and in the throats in 11% of patients during the first week of illness, and in the stools of 57% of cases and in no cases in the throat during the second week. It is seen that the virus is eliminated with greater frequency and for longer periods in the stools than in the material from the pharynx. The bacteriological evidence is strongly suggestive that during an epidemic the faeces are by far the largest source of virus available for distribution, but on the other hand, the virus is transmitted mainly by droplet infection.

In the preparalytic stage of the infection, gastro-intestinal symptoms due to a non-specific enteritis may be present.

that mental fatigue and worry are other predisposing factors. It was found that four of our cases in the age group 0-5 years gave a definite history of injury one to seven days before the onset of paralysis. Of the 5-15 age group, one gave a history of appendicitis with drainage twelve days previously, and here the paralysis affected both legs, the abdominal muscles and the erector spinae. Three cases over 15 years of age admitted to strenuous exercise twenty-four hours before the onset of paralysis of both legs.

Investigations designed to show transmission of the virus by water, food, milk, sewage, dust, flies, mice and other vermin have been numerous, but so far no definite conclusions have been reached. During epidemics, flies have been found carrying the virus in and around poliomyelitis districts, but there is no evidence of its multiplication. Flies may be simply accidental reservoirs of the virus and may be of no importance in the spread of poliomyelitis, or they may be an important link in the epidemiological chain of events leading to poliomyelitis epidemics. The common domestic and farm animals and birds have all been considered insusceptible to the human virus, and there is no evidence of the virus being carried by them, nor has it ever been discovered in their faeces. The poliomyelitis virus is not found in certain fish from water polluted with infected faeces, nor in the faeces of swallows feeding on flies in the neighbourhood of infected sewage.

Manning (1912) was of the opinion that the bed bug *Cimex lectularius* could transmit poliomyelitis from man to man, but further investigations have not supported this view.

Direct transmission through the skin may occur, and crowded swimming-pools, beaches and camps are other possible sources of danger.

Since the first cases of muscular paralysis occurring in teething children were described by Colmer (1843) and by Fliess (1849), a large number of workers have investigated this problem, but so far no definite relationship has been established. Cameron (1947) could find no evidence that the exposed dental pulp provided an important point of entry for the virus. Dental extraction should be limited, however,

excreting the virus, or that a proliferation had occurred outside the body. There is, however, no known virus reservoir apart from man. As the virus of poliomyelitis is present in the faeces of typical and abortive cases during the acute and convalescent stages of the infection, and as it has been recovered from the sewage of towns when cases were occurring in that area, the possibility of water from contaminated rivers and swimming-pools being responsible for the spread of the disease must be considered. The general opinion, however, appears to be that there is no convincing evidence that the large epidemics in the United States and America are related to the water supply. The possibility of faecal contamination of railway tracks in the proximity of the town may be a real source of the dissemination of the poliomyelitis virus.

Although the attack rates in rural districts are often higher than in towns, the reverse is sometimes true in some countries, and the fact that outbreaks do not recur in the same area in consecutive years suggests a widespread latent immunization to the virus.

There seems to be little doubt that epidemics occur in the countries with the highest standards of living. It may be that in primitive civilizations infants are repeatedly exposed to infection, and thus acquire a lasting immunity, but if the sanitation is improved then epidemics will occur. The increase in the number and size of the epidemics, the change in the incidence to the older age groups and the greater mortality in adults may be due to better modern sanitation.

The great rarity of infection amongst those nursing cases of paralytic poliomyelitis, despite the presence of the virus in the faeces, is in direct contrast to that which occurs in cases of typhoid and dysentery.

In 1880 Bull described a case of poliomyelitis following exposure to severe cold and wet, and Dornedden (1933) expressed the opinion that preliminary catarrh or injuries were predominant factors in paving the way for the virus. Experimentally it has also been shown that warm weather, body chilling and fatigue affect adversely the development and severity of the paralysis. Clinically it has been concluded

or no paralysis. The majority occurred within the first week, this agreeing with the observations of other workers. Zintek (1947) carried out weekly throat washings and faeces examinations in a family which had been in close contact with a case of poliomyelitis, and the virus was demonstrated at least once during the period of observation in the father, mother and three children.

Even although new cases in a family continue to appear for several weeks, all may possibly have been affected at the same time.

The incubation period usually lasts from seven to twelve days from the time of exposure, but it may vary between three and forty-five days, as apparently healthy children have been known to carry the virus for a month before they had shown clinical signs of the disease. The period of greatest infectivity seems to be during the latter part of the incubation period and the first week of the acute illness, and all children and adults who are contacts or who appear to be indisposed during this period should be most carefully watched.

Batten (1910) stated that in one of his cases the clinical history was sufficiently definite to conclude that it was a case of intra-uterine infection. Blair and Robertson (1944) reported on six cases of poliomyelitis occurring in pregnant women and indicated that the child in utero was safe from even a severe maternal attack. From this and other investigations, the present accepted view is that the unborn child is immune to the disease. Gunewardene (1918), Cislak (1934) and Biermann and Piszczek (1944) described three cases in which the infants were 12, 14 and 11 days old respectively. In a survey of nearly 18,000 cases carried out in 1939, it was found that there were only 22 babies affected during the first month of life.

It is now generally accepted that the majority of children are relatively immune during the first year of life, even when in close contact with a definite case, and this has been attributed to a passive immunity derived from the mother. If a baby does develop the disease, it is usually of a very mild nature. In our series of cases, ten were under one year of age, seven being females and three males. One or both legs were

during an epidemic to urgent cases, because like tonsillectomy and adenoidectomy, trauma is inflicted locally and peripheral nerve endings are exposed, and these may act as direct portals of entry.

The seasonal incidence of the disease in Great Britain seems to coincide with the appearance of soft fruits in the shops, and the disease may therefore be spread by the ingestion of unwashed and diseased fruits and vegetables affected by the neurotropic virus. The fruit may be affected by the virus from a sprayed droplet infection, contaminated hands, or directly from faecal-feeding flies or from dust.

It has been suggested that deficiencies in the diet producing a relative excess or lack of vitamin B complex may be the cause of the difference of susceptibility of the cases.

Anderson (1947) believes that those who become paralysed are physiologically so constituted that there is destruction of the nerve cell, and this may explain the unusual susceptibility of some families.

Packard (1879) reported on a case where a brother aged $2\frac{1}{2}$ years and his sister aged $1\frac{1}{2}$ years were affected simultaneously, whilst Pasteur (1896-97) described an epidemic occurring in seven children of the same family within a period of ten days.

In one report of 900 cases, there were only 23 instances of two cases in the same family, and in the majority of these, the second had occurred within ten days of the onset of the first.

In the outbreak among the Eskimos in 1948-49, multiple cases in families were common, 12 out of 25 families having more than one case.

Family outbreaks of poliomyelitis are fairly common and although often only one child becomes paralysed, the others may have minor illnesses compatible with abortive poliomyelitis and the virus can usually be isolated from them. In our series of 500 cases, 22 occurred where a definite relationship could be established of contact with another case of poliomyelitis in the same family. The first case usually showed definite clinical signs with paralysis, whilst the other relative or relatives showed only the initial clinical signs with minimal

It will be noted that approximately two-thirds of the cases were under 15 years of age and approximately one-third under 5 years.

It is also important to note that patients in rural districts have a greater tendency to be drawn from the older age groups than those in urban areas. It is of interest that the oldest recorded case is that of a man aged 68 years.

It has been reported that the largest proportion of abortive cases are in the latter age groups, and the maximum number of non-paralytic cases appears to be in the 5-14 age group.

Horstmann (1946) observed that the majority of the paralytic cases were in the 0-4 age group and other observers have stated that the great vascularity of the spinal cord about the second year of life may contribute to the susceptibility at this age. In 1948 Bradley and Gale made the important observation that the severity of the disease was much greater in the later age groups, in other words, the tendency to severe paralysis increases with advancing years.

It should be noted that in the earlier epidemics the limbs and trunk appear to be mainly involved, but in the more recent epidemics the incidence of brain stem involvement has been particularly high.

There seems to be a slight preponderance of males over females and the ratio usually quoted is 5:4. This is especially true in patients under 5 years of age. It therefore follows that there is an excess of females if the patients are over 5 years of age.

The table overleaf shows the number of male and female cases at the various ages in our series.

Extensive research in this and other countries has failed to show any association between the rate of the attack and the different levels of social status within the population.

McCloskey (1950) reported upon 340 cases of poliomyelitis and Geffen (1950) on 182 cases which were associated with inoculations in 24 and 30 cases respectively.

It seems conclusive that children under 3 years of age are made more vulnerable to infection by the poliomyelitis virus after the administration of pertussis vaccine or combined pertussis and diphtheria vaccine, especially from seven

usually affected but in two cases the left arm was involved. One of these also had a paralysis of the seventh cranial nerve.

When poliomyelitis is a sporadic endemic disease and during the first epidemics experienced by a country, approximately 90% of the paralytic cases occur in children under 5 years of age. This was clearly demonstrated in the Malta epidemic (1945) and those which occurred in Japan in 1938 and 1940. The age incidence varies greatly, however, in different epidemics as is shown by the following figures: Craster (1916) reported that 86% of 1,360 cases were under 5 years of age, and Blencke (1933) found that 70.3% of 1,695 cases were also in this group. In later epidemics the proportion of cases occurring in the later age groups increases with each epidemic, about 50% occurring in the 0-5 age group and about 80% occurring in the 0-10 group. For example, in 1907 in Massachusetts, only 7% of cases were over 15 years of age, but in 1945 this had increased to 25%. In Scandinavia in 1934, 20% of cases were over 15 years of age, but in 1944 the percentage had increased to 53. In the 1947 epidemic in the British Isles, approximately one-third of the cases were in each of the age groups 0-5, 5-15 and over 15 years. Horton and Rubinstein (1948) attributed this change in the age distribution to an alteration in the age of the population and to an increase in the number of non-paralytic cases reported. It is nevertheless now generally agreed that the age incidence has altered greatly during the last few decades and that except during epidemics cases are rare in the adult population. The following table shows the age incidence in our series of five hundred cases.

TABLE II

Age	No	Age	No	Age	No	Age	No
0-1	14	5-6	20	10-15	48	35-40	38
1-2	29	6-7	20	15-20	42	40-45	28
2-3	56	7-8	20	20-25	37	45-50	3
3-4	42	8-9	20	25-30	29	50-60	1
4-5	30	9-10	22			60-70	1

It will be noted that approximately two-thirds of the cases were under 15 years of age and approximately one-third under 5 years.

It is also important to note that patients in rural districts have a greater tendency to be drawn from the older age groups than those in urban areas. It is of interest that the oldest recorded case is that of a man aged 68 years.

It has been reported that the largest proportion of abortive cases are in the latter age groups, and the maximum number of non-paralytic cases appears to be in the 5-14 age group.

Horstmann (1946) observed that the majority of the paralytic cases were in the 0-4 age group and other observers have stated that the great vascularity of the spinal cord about the second year of life may contribute to the susceptibility at this age. In 1948 Bradley and Gale made the important observation that the severity of the disease was much greater in the later age groups, in other words, the tendency to severe paralysis increases with advancing years.

It should be noted that in the earlier epidemics the limbs and trunk appear to be mainly involved, but in the more recent epidemics the incidence of brain stem involvement has been particularly high.

There seems to be a slight preponderance of males over females and the ratio usually quoted is 5:4. This is especially true in patients under 5 years of age. It therefore follows that there is an excess of females if the patients are over 5 years of age.

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It seems conclusive that children under 3 years of age are made more vulnerable to infection by the poliomyelitis virus after the administration of pertussis vaccine or combined pertussis and diphtheria vaccine, especially from seven

to sixteen days after injection. The vulnerability seems definitely localized to the most recently injected limb, and when the virus reaches the central nervous system during this period, severe local paralysis results. In St. Pancras, of 19 children under 5 years of age who had developed poliomyelitis, 6 had been immunized within the previous fortnight, and the paralysis was limited to the left arm into which an intramuscular injection with the combined vaccine had been given. It seems likely that an inoculation with an antigen during the early stages of poliomyelitis infection does increase the incidence of paralysis as well as determining the site, but no

TABLE III

Age	Sex M F		Age	Sex M F	
0-1	6	8	10-15	23	25
1-2	16	13	15-20	22	20
2-3	32	24	20-25	15	22
3-4	20	22	25-30	14	15
4-5	20	10	35-40	23	15
5-6	11	9	40-45	14	14
6-7	9	11	45-50	1	2
7-8	11	9	50-60	—	1
8-9	9	11	60-70	1	—
9-10	10	12			

definite decision is justified without further extensive study. The significant finding which we have also found is that there is clearly an association between the site of the last injection and the site and degree of subsequent paralysis.

In uninoculated patients the age of the child is important in determining the site and the severity of the paralysis in poliomyelitis, because in younger children there is a tendency for the lower limbs to be affected. In inoculated children, on the other hand, whatever the age of the patient, the injections into the arm were associated with an increased frequency and clinical severity of upper limb paralysis. In most of the cases the interval between the date of the inoculation and the recorded date of the onset of the symptoms

was between eight and seventeen days which roughly corresponds with the incubation period of poliomyelitis. A differential diagnosis must, however, be made as to whether these are cases of brachial neuritis such as occur after other injections or whether they are true cases of poliomyelitis. The most likely explanation is that the local tissue damage caused by the injection has affected the nerve supplying this area, by some kind of local axon reflex, and it has been suggested that a state of hypersensitivity to paralytic poliomyelitis develops about the seventh day after inoculation. It is localized to the injected limb and reaches its height about the eleventh day. The injection would have very much the same effect as muscular effort in converting a silent infection into a paralytic one. Another explanation is that in a patient suffering from a non-paralytic infection, the circulating virus is arrested and concentrated in the tissues damaged by the vaccine. It then travels by the peripheral nerves or some other channel to the corresponding areas in the cord. Bousfield (1950) suggested that the use of P.T.A.P. antigen subcutaneously solves the problem of the current diphtheria immunization.

In spite of the most extensive research, it must be admitted that the epidemiology of poliomyelitis is still undetermined. This is due to the following main factors

- (1) The route of the infection is unknown
- (2) The manner of transmission is unknown
- (3) The uncertainty of making an early and definite diagnosis.
- (4) The absence of any specific test for susceptible persons, sub-clinical and abortive cases
- (5) There is no suitable test for past infection

BIBLIOGRAPHY

- ANDERSON, G. W. (1947) *J. Lancet*, 67, 1013
ANDERSON, G. W., *et al* (1950) *Ann. Otol. (St. Louis)*, 59, 602
ANDERSON, J. A. (1945) *J. Pediat.*, 27, 68
ASIWORTH, P. G. (1948) *M. J. Aust.*, 1, 793
AYCOCK, W. L., and LUTHER, F. M. (1929) *New Eng. J. Med.*, 200, 164

- BATTEN, F E (1910) *Brain*, 33, 149.
- BATTEN, F E (1912). 17th Int. Congr Med London Sec 10, Dis Chil, Part II, p 145
- BARLOW, W H (1878) *Liverp Manch med Surg Rep*, 6, 1.
- BIERMANN, A. H., and PISZCZEK, E. A (1944) *J. Amer med Ass.*, 124, 296
- BRYANT, W S (1910) *N. Y med J.*, 92, 1215
- BRADLEY, W H., and GALE, A. H (1948) *Publ Hlth Rep. (Wash)*, 63, 397
- BROWN, W G S (1932) *J Neurol Psychopath*, 12, 309.
- BRYANT, W S (1910) *N. Y med J.*, 92, 1215
- CASEY, A E, et al (1945) *J Amer med Ass*, 129, 1141.
- CISLAGHI, F (1934) *Pediatrics (Napoli)*, 42, 1456.
- COLMER, G (1843) *Am J med Sci*, 5, 248
- CRASPER, C V (1916) *Trans Amer Ass Study Prev. Inf. Mortal*, 7, 187
- CUNNING, D S. (1946) *Ann Otol (St. Louis)*, 55, 583
- FISCHER, A E, and STILLERMAN, M. (1941) *Amer J Dis Child*, 61, 305
- FLEXNER, S, and LEWIS, P A (1910) *J. Amer. med Ass*, 54, 1140
- FLIESS, M (1849) *J Kinderk*, 13, 39.
- GEFFEN, D H *Med Offr*, 83, 137
- GUNWARDENE, T H (1918) *Lancet*, 2, 847
- HORSTMANN, P (1946) *Acta med Scand*, 124, 482
- HORSTMANN, D M, et al (1944) *J. Amer med. Ass*, 126, 1061.
- HORTON, R J M, and RUBINSTEIN, A. D (1948) *New Eng J. Med*, 239, 169.
- MCNULTY, A (1938) *Lancet*, 1, 1288
- PACKARD, F A. (1879) *J nerv ment. Dis*, 26, 210
- PASTEUR, W. (1896) *Trans. clin Soc, Lond*, 30, 143
- PAUL, J. R. (1944) *Yale J. Biol Med*, 16, 462.

- PAUL, J. R. (1947) *Amer J Hyg*, **45**, 206
 ROBERTSON, E. G. (1940) *Med J Aust*, **1**, 156
 SEIGEL, M., *et al* (1951) *J Pediatr*, **38**, 537
 SIMPSON W. (1975) *Amer J Hyg*, **102**, 210

8, 653.

451

- BATTEN, F E (1910) *Brain*, 33, 149.
BATTEN, F E (1912) *17th Int Congr Med London Sec 10, Dis. Child*,
Part II, p 145
BARLOW, W H (1878) *Liverp Manch med Surg Rep.*, III, 1.
BIERMANN, A H, and PISZCZEK, E A (1944) *J. Amer. med Ass.*,
124, 296.
BROWN, W G S (1932). *J Neurol. Psychopath.*, 12, 309
BRYANT, W S (1910) *N.Y. med J.*, 92, 1215.
BRYANT, W S (1920) *Lancet*, I, 567
CUNNINGHAM, D S (1946) *Ann Otol (St Louis)*, 55, 583
FISCHER, A E, and STILLERMAN, M (1941) *Amer. J Dis Child*, 61,
305.
FLEXNER, S, and LEWIS, P A (1910) *J Amer med Ass.*, 54, 1140
FLIESS, M. (1849) *J Kinderk.*, 13, 39.
GEFFEN, D H *Med Offr.*, 83, 137.
HOWE, H A, and BODLEY, C (1942) *Amer. J Hyg.*, 35, 19
MCNULTY, A (1938) *Lancet*, I, 1288
MORSE, R (1942) *Amer. J. Hyg.*, 35, 26, 210
MORSE, R (1942) *Amer. J. Hyg.*, 35, 30, 143.
MORSE, R (1942) *Amer. J. Hyg.*, 35, 462.

embryonic brain. All culture preparations are made from the faeces or infected nervous tissue, and in fatal cases the virus can be recovered in 50% of the patients from the brain stem and spinal cord. In man, however, it has never been found in the cerebrospinal fluid and only on one occasion in the blood of clinical cases.

Investigations have shown that poliomyelitis is a symptom complex, produced by at least three groups of human viruses which are antigenically distinct. There may be several other antigenically distinct strains of poliomyelitis virus, because all material from patients dying from this disease does not yield positive results on inoculation into monkeys.

Levaditi *et al.* (1936) determined the size of the causative virus by ultrafiltration through collodion membranes, and estimated that its diameter was between eight and thirteen millimicra (μ).

In the Lansing type, the virus can be transmitted from man to monkeys, and then to cotton rats and white mice. Jungeblut and Dalldorf (1943) isolated from the house mouse a virus which was associated with an outbreak of human poliomyelitis, and a similar virus has been isolated from the brain stem of a fatal human case. This virus produced paralysis in albino mice, cotton rats and hamsters. Six of these rodent-adapted strains have been described and isolated from patients in America and the Middle East, whilst virucidal antibodies to these viruses have been found in Europe, America, Asia and Africa.

The second group of viruses includes the Amengo-encephalitis virus from Uganda, the E.M.C (encephalomyocarditis) virus isolated from a monkey in California, and the M.M. and Columbia S K. viruses which originate from rodents. The outbreak of poliomyelitis in American troops in the South-West Pacific area was thought to have been due to one of these Columbia S K group of viruses. This second group of viruses can be transmitted to man, monkeys and rodents, and it can be transmitted to man.

CHAPTER II

PATHOLOGY

DISEASES closely resembling poliomyelitis in clinical features and pathology occur naturally in mice and pigs, but although the viruses are of a similar size, they are quite distinct from the human strains. There are also numerous strains of virus capable of producing poliomyelitis or poliomyelitis-like symptoms, but their exact relationship to the disease has not yet been clearly defined.

Poliomyelitis is caused by a neurotropic virus which acts directly on the nerve cells. The brunt of the attack appears to fall on the ventral horns of the cervical and lumbar enlargements of the spinal cord, but the nerve cells in the cerebrum, cerebellum, brain stem and automatic nervous system may also be affected. In some cases the virus is destroyed before the nerve cell is damaged to any great extent, and then only a temporary paralysis or paresis results. In other cases it continues to multiply during its period of activity, resulting in a total destruction of the motor cell, and a varying degree of permanent paralysis.

In 1908 Landsteiner reported on the first successful transmission of poliomyelitis to the monkey, and in 1909 Flexner and Lewis inoculated a series of monkeys with the virus of poliomyelitis from an emulsion of the spinal cord of children who had died from that disease. They also demonstrated that the inoculated monkeys developed a definite paralysis whether the intraperitoneal, intravascular or intraneural route was employed. Further investigations by these two workers in 1910 also showed that active immunization and passive serum protection could be given to the monkeys. Another equally important point shown was that the monkeys cannot contract the disease directly from each other or from human beings.

The neurotropic virus appears to be exclusive to man, anthropoid apes and monkeys, but it is contagious only to man, and it must be cultivated in tissue cultures of human

spinal cord on the day before paralysis; that is, at the time when histological lesions were first demonstrable. The concentration of the virus was also shown to reach its maximum in the next day or two and then to fall rapidly. They found that the concentration varied with the stage of the disease rather than with the extent of the paralysis. The antibodies, however, did not appear until approximately fourteen days after the onset of the paralysis, but they persisted for a period of four to five months.

Howe and Bodian (1941) demonstrated that the distribution of the lesions in the human brain was comparable with that found in monkeys inoculated by the non-nasal routes, thus showing that the virus travelled in the central nervous system by the fibre pathways. Experimentally it has also been demonstrated that following intracerebral inoculation and transection of the cord, the segment of the cord distal to the level of the transection did not show any inflammatory changes, nor could the virus be isolated from it. As the blood supply to the cord remained intact and the circulation of the cerebrospinal fluid unimpaired, the conclusion to be drawn appears to be that neither the blood nor the cerebrospinal fluid plays any important part in the dissemination of the virus.

In experimental infections the inflammation is most marked in the cervical and lumbar enlargements, irrespective of the route by which the experimental inoculation is given. In one experiment the virus was injected into the sciatic nerve and it was noted that the lesion appeared first of all in the lumbar enlargement of the same side, it then spread upwards to the corresponding centre in the opposite cerebral cortex, and it was detected there before it appeared in the cervical enlargement.

All the evidence available appears to favour the invasion of the nervous system along the axonal pathways of the peripheral nerves and its further transmission throughout the brain and cord by the various nervous tracts. According to Howe and Bodian (1942), the transmission of the virus in the nerve fibres in rhesus monkeys is at the rate of two to four millimetres per hour.

The third group of viruses isolated in America and Western Europe can be transmitted only to suckling mice or hamsters. The Coxsackie viruses may be isolated from human faeces and will infect suckling mice, causing paralysis due to lesions of muscles rather than of brain and spinal cord. It has also been stated that the Coxsackie virus produces non-paralytic poliomyelitis.

The poliomyelitis virus can be preserved in a frozen or dried state, but it is destroyed by some weak disinfectants and exposure to temperatures of 45° to 50° C. for half an hour. It is readily destroyed by oxidizing agents, e.g. hydrogen peroxide and potassium permanganate and by ultra-violet rays, but the latter cannot be relied upon to disinfect satisfactorily. It can remain virulent in drinking water, milk and an aqueous suspension of faeces for approximately one month. Ordinary amounts of chlorine used for water purification have been found to be insufficient to kill the virus, more especially if organic matter is present.

Experimental evidence suggests that some strains of the virus seem to enter the body more easily through one route, whilst others will choose another.

In the rhesus monkey, experimental studies have established that the nasopharyngeal route is the common one and histological changes are seen to accompany the spread of the virus from the olfactory bulbs via the thalamic region to the spinal cord. It is interesting to note that sectioning of the olfactory nerve in monkeys can definitely prevent infection by the intranasal route. Faber *et al.* (1947) demonstrated that if the virus be applied to the central end of the divided branch of the trigeminal nerve, it will travel centripetally to the corresponding semilunar ganglion within three days. Centrifugal spread to the nasopharyngeal surface was also demonstrated by the detection of the virus in the nasopharyngeal washings on the third and fourth days, whilst the virus was found only in the stools on the fourth day, suggesting that it had reached the intestines by swallowing.

Bodian and Cumberland (1947), using the Lansing mouse-adapted strain of virus in rhesus monkeys, showed that after intracerebral inoculations the virus could be found in the

months of the original attack could be regarded as ■ recrudescence of the original infection.

The incidence of second attacks has been estimated at about 1/10,000 cases of recovered poliomyelitis patients

Second attacks may occasionally occur which are due to the virus entering the body through a different portal. Fischer and Stillerman (1938) pointed out that a non-paralytic attack may precede or follow a paralytic attack.

It is only right to point out that atypical second attacks may have occurred but have been missed, as the only symptoms may be fever, vomiting, sore throat, meningeal signs and muscle weakness and spasm

Recently antibodies have been demonstrated in the nasopharynx but not so far in the stools. This may be the explanation for the increasing difficulty in isolating the virus from the nasopharynx after the first week from the onset of the disease, and the persistence of the virus in the stools for several weeks

We now turn our attention to the subject of morbid anatomy and histology. Although the spinal cord bears the brunt of the attack, the whole central nervous system can be affected to a greater or lesser extent

The virus of poliomyelitis has been observed to produce lesions in the cerebral cortex, the basal ganglia and the thalamic and hypothalamic nuclei. The mid-brain, cerebellum, pons, medulla oblongata, spinal cord and the autonomic nervous system are also involved. The lesions in the cerebral cortex are confined mainly to the motor and pre-motor areas, as the virus appears to have a special affinity for the large pyramidal cells in these regions, whilst in the cerebellum they are mainly confined to the roof nuclei, the vestibular nuclei and the reticular formations

The reaction of the tissues to the infection varies greatly in its intensity and it would appear to depend mainly upon the dose, type and virulence of the strains of the virus. The portal of entry, the site of the infection in the nervous tissue and the resistance of the patient may also be important factors.

The histological changes are always found to be much

It is possible that the strains of the poliomyelitis virus are immunologically different, and that each strain affords protection only against itself. In 1937 Flexner showed that monkeys could be readily reinfected by the virus used in the first instance, as well as by a different strain of virus. It has also been shown experimentally that chimpanzees, when given by mouth a strain of virus of which they have previously been carriers, are found to be completely immune from infection by it, but if different strains are used, they can be reinfected.

It was formerly taught that an attack of poliomyelitis conferred a life-long immunity by the production of antibodies, but although second attacks of the disease are rare, they are not unknown. Bridge *et al* (1946), reviewing the literature, found reports of only thirty-one instances of second attacks and Alves and Pugh (1947) added another. One of our cases gave a history of an attack at the age of 6 months when the erector spinae group of muscles was affected. The second attack was at the age of 33 years and both legs were then involved.

Neutralizing antibodies may be detected in the blood serum in 60% to 70% of the normal adult population which points to the fact that they have suffered from a sub-clinical or abortive attack, or that they have become immune as the result of repeated exposure to a less virulent form of the virus without at any time showing signs of illness. Although a patient with paralytic poliomyelitis may develop antibodies against his own strain of virus which may persist throughout his life, he may still be susceptible to an immunologically different strain of which multiple varieties have been demonstrated, e.g. Lansing, S.K., M.M. strains and Theiler's virus. These second attacks may occur in abortive forms during an epidemic, but it is doubtful whether these sub-clinical infections are necessary to maintain immunity. There appears to be no doubt that following on a severe attack with extensive paralysis, the immunity extends over a number of years. Still (1930) stated that in his opinion two years was the probable maximum duration of immunity resulting from an acute attack, and that any attack which occurred within four

months of the original attack could be regarded as a recrudescence of the original infection

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The histological changes are always found to be much

more widespread than the clinical evidence would indicate. A very severe inflammatory reaction may be present but without any evidence of correspondingly severe damage to the nerve cells. On the other hand, we may find an extensive destruction of the nerve cells without any accompanying oedema or marked vascular changes, and the inflammatory reaction may be minimal or absent.

Of course, the pathological findings are greatly altered or modified according to the length of time which has elapsed between the initial onset and the death of the patient. In fulminating cases, involving the cerebral cortex, mid-brain, basal ganglia and cerebellum, the nerve cells may show practically no change. In cases in which the brain stem was involved and which lived for twenty-four to forty-eight hours, there are usually a large number of neuronal lesions in the medulla. These are most marked in the region of the nucleus ambiguus, the dorsal nuclei of the vagus nerve and the nucleus solitarius. In less acute cases, where death has occurred after the onset of the paralysis, the whole of the central nervous system is found to be slightly or moderately oedematous, the brain stem congested and the blood-vessels in the anterior horns of the spinal cord distended.

In these less acute cases, if lesions are looked for in other parts of the brain, they will be found fairly frequently in the substantia reticularis, in the floor of the fourth ventricle, in the substantia nigra and around the aqueduct of Sylvius. Less frequent and less severe lesions may be detected in the corpus striatum, the globus pallidus and the hypothalamic nuclei.

It is important to try to correlate these pathological findings in the central nervous system with those of the clinician, and the following observations have been recorded.

Softening around the basal nuclei with a definite loss of the ganglion cells is a common finding in cases which have suffered from stupor, coma or disorientation before death. Hysterical and psychological manifestations are also attributed to lesions of the hypothalamic centres. If there has been marked dysphagia and dysarthria, the lesions are usually found around the nucleus ambiguus.

Sheinker (1947) reported on a pronounced inflammatory and degenerative tissue reaction in the region of the vagal nuclei, and this appeared to substantiate further the theory of the spread of the virus from the alimentary tract to the central nervous system by means of the afferent and efferent fibres of the vagus nerve. Certain other observers have pointed out that the nuclei of the seventh and twelfth cranial nerves are also often involved. The ganglia of the fifth, ninth and tenth cranial nerves which carry afferent impulses from the pharynx have also shown signs of invasion in a large number of human cases.

If in the human species the olfactory bulbs and tracts are examined histologically, they are almost invariably found to be normal. In cases which have died following manifestation of bulbar poliomyelitis, the spinal ganglia may also be found to be of normal appearance.

If we now turn our attention to the spinal cord, we find that the initial attack may be focused upon certain groups of motor neurones, or it may be spread amongst the individual nerve cells in the anterior horns. An important point to note is that if the attack is bilateral, it is usually of an asymmetrical nature.

If the spinal cord is sectioned and examined at different levels, it will be found that certain areas showing marked involvement may be interspersed with other areas of apparently normal tissue.

A most important experiment was carried out by Bodian and Howe in 1941 as the result of which they concluded that the production of the inflammatory reaction depends on the presence of living nerve cells. Before inoculating the virus, they allowed the nerve cells on one side of the cord to degenerate and they found that no inflammatory reaction had been produced on that side. When they examined the opposite side, they found that a nerve cell degeneration and a typical inflammatory reaction had occurred.

The typical lesions are most pronounced in the brain stem and anterior horns of the spinal cord at levels corresponding to the paralysed muscle group, but they occur only when the virus has reached the nerve cells via their axons.

The changes which are about to be described usually precede the onset of the paralysis by at least twenty-four hours. According to Bodian (1948) the axons undergo Wallerian degeneration between the third and fourth days after the destruction of their cell bodies. It has been pointed out that this is the usual time for a secondary degeneration to occur, and it is therefore thought to indicate that the primary damage to the motor neurone is in the cell itself. The nuclear



FIG. 2.—CELLULAR INFILTRATION OF HUMAN SPINAL CORD

[Reproduced by permission, Jennings, G. H., Hamilton-Paterson, H. L., and MacCallum, F. O. (1949), *Brit med J*, 2, 206]

changes, however, occur only after the onset of chromatolysis in the cytoplasm. If the sections are carefully examined, varying degrees of chromatolysis will be seen, and if the degenerative changes in the cell are not too far advanced, they are reversible, and a complete recovery of the muscle is possible.

It has been found that a full recovery of the nerve cells usually occurs within two months but that the inflammatory reaction takes longer to subside. An apparent recovery of

the nerve cells ■ not necessarily accompanied by an immediate and complete recovery of the paralysis

A partial or complete destruction of the nerve fibres in the anterior commissure may also be found. A similar condition may be present in the anterior nerve roots and muscular branches of the peripheral nerves.

Polymorphonuclear leucocytes are seen outside the blood-vessels within a few hours of the onset, but they are later



FIG. 3—PERIVASCULAR CUFFING

[Reproduced by permission, Jennings, G. H., Hamilton-Paterson, H. L., and MacCallum, F. (1949), *British Medical Journal*, 2, 206.]

replaced by lymphocytes and plasma cells. It will be noted, however, that there is no fibrinous exudate. The blood-vessel walls are thickened and the perivascular sheaths become distended with lymphocytes and other mononuclear cells. Some of the smaller blood-vessels are thrombosed and small haemorrhages may also be present. These appearances are most marked in the grey matter of the spinal cord.

The extent of the perivascular infiltration of the small mononuclear cells appears to be proportional to the vas-

cularity of the area, and it is most evident in the floor of the fourth ventricle and in the cervical and lumbar enlargements. Mayr (1932) found that the spinal cord was most frequently affected below the fourth lumbar segment. This infiltration may also be evident in the anterior fissure where the larger veins leave the spinal cord.

A marked increase in the number and size of the microglia is also noticed and their nuclei become markedly elongated. The oligodendroglia in the proximity of the focus undergo acute swelling, but those in the immediate neighbourhood of the nerve cells die. The astrocytes frequently lose their processes, become markedly swollen and finally necrotic.

Infection may ascend or descend in the grey matter of the spinal cord and in this way several segments may be involved. The chief method of spread appears to be along the nerve fibres, e.g. Clarke's column, but probably both sensory and motor fibres can transmit the virus.

It has been found experimentally that after inoculation of a mixed nerve, the first lesions to be found are those in the spinal ganglia. At post-mortem the posterior root ganglia show signs of invasion, particularly those in the segments where the anterior and lateral horns are involved.

Neumann (1950) stated that he thought that the pain complained of was probably due to the involvement of the posterior roots and their spinal ganglia. The posterior horns of the grey matter may also be affected in many cases, and this will cause motor dysfunction by interference with the spinal reflex arc.

Other observers have stated that the most commonly affected parts are those in the regions containing the cell bodies of the internuncial neurones. Attempts have accordingly been made to explain the muscle spasm, alienation and muscular inco-ordination by these lesions.

In a few cases the inflammatory process extends into the lateral columns of the spinal cord, whilst in the rare cases in which there is an impaired appreciation of the pain and temperature senses, the spino-thalamic tracts are found to be involved.

Two other rare phenomena may also be observed. In the

first of these, the damage to the motor cells in the brain or to the pyramidal tracts leads to a spastic paralysis in the related muscles, and secondly, if the cervical region is involved, there is a spastic paraplegia associated with muscular wasting of the arms

Fairly recently it has been postulated that the virus may also extend along the sympathetic fibres to their ganglia. Clinically this has been demonstrated by cyanosis, sweating and disturbances of micturition which may develop before the onset of the paralysis. Also at post-mortem the virus may be found in the sympathetic ganglia or they may show evidence of having been invaded. Some of the ganglion cells are swollen and have clear cytoplasm, but others may be shrunken and no nucleus is seen. The evidence therefore strongly suggests that the virus usually travels by the visceral afferents.

Nelson (1946) stated that in his opinion the determining factor in the distribution of the paralysis was an alteration in the circulation of the blood in the spinal cord. An extensive inflammatory oedema may also press on the lumen of the vessels and so cut off the circulation. The damaging effect on the cells and the extent of the resulting paralysis may therefore be due to

- (1) Direct pressure from the haemorrhage
- (2) Direct pressure from the inflammatory oedema
- (3) Anaemia due to constriction or thrombosis of the vessels
- (4) Direct toxic action of the virus

The first two of these causes usually produce enough pressure on the anterior horn cells to cause only temporary inhibition of the function. The last of these causes may also produce a temporary inhibition of the function of the anterior horn cells or it may completely destroy them.

As mentioned above, some of the nerve cells are destroyed by the direct action of the virus during the acute stage, but a large number recover their normal appearance in a few days or weeks. This recovery may be the primary factor in the early restoration of muscle function, and lead to a great

reduction in the ultimate paralysis. A part of the recovery of muscle power may also be explained by the development of new neuromotor patterns involving the use of the remaining motor cells which have not undergone irreversible degenerative changes.

Regressive changes may take the form of sclerosis or vacuolation of the cells. Degenerative alterations in the neurofibrils and disintegration of the cell walls and nuclei may also be seen. The presence of abnormal or increased lipid deposits in some of the cells may also be observed.

We must now consider cases in which death has occurred during the first few weeks. Here we find an irregular congestion of the leptomeninges over the affected area of the spinal cord and some haemorrhage into the ventral horns. These changes are found to be most marked in the cervical and lumbar enlargements, and characteristically they are asymmetrical.

Examination of the anterior nerve roots shows that they have become atrophied and the anterior horn cells appear to be diminished in number. Some of these cells are seen to be shrunken, distorted or degenerated. There is also an increase in the neuroglia and a coarsening of the delicate reticulum which the fibres usually form.

Lastly we come to the changes which are usually observed in cases which have died several months or years after the acute onset. In these cases the anterior horn cells are found to be pale, shrunken and sclerotic, and when they are examined microscopically the anterior horn cells may be found to be absent. The first description of this type of case was given by Taylor (1878-79). This was in a child of three years who had had poliomyelitis affecting the left leg at the age of fifteen months. The cord was found to show marked atrophy of the left side of the lumbar enlargement and this was specially noted in the region of the anterior horn.

Let us now review the pathological changes which may also be observed in the pharyngeal mucous membrane, skin, muscles, bones, ligaments, lymphatic glands and other tissues and organs.

As early as 1912, Neustaedter described the pathological

changes which he had observed in the pharyngeal mucosa. He found that it looked anaemic and that it had a glistening and oedematous appearance. In some cases, a serous frothy exudate was present and this persisted for some weeks after the onset of the paralysis

The skin should next be examined and it is often found to be hypersensitive and tender during the acute phase. This, however, may be missed, as it lasts in the majority of cases for only a few days. In rare cases it has been known to persist for months. The real cause has not been definitely established, but it has been attributed to the affection of the posterior nerve roots and the posterior columns of the spinal cord. At a later stage the skin may become less elastic and thickened, with disappearance of the subcutaneous fat

As there is frequently a marked disturbance of the circulation in the involved extremity, vasomotor changes should be looked for in the convalescent and residual stages of the disease. It usually manifests itself in blueness and coldness of the limb and perspiration may be excessive. Trophic changes are very commonly present in the late convalescent and residual stages and the patient may suffer greatly from chilblains and ulcers

The skeletal muscle fibres obey the "all or none" law, so that the destruction of a single anterior horn cell results in paralysis of a minute portion of the muscle. It therefore follows that the more nerve cells involved, the greater will be the weakness of the muscle. If all the nerve cells are destroyed, the related muscles will be completely paralysed, resulting in atrophy and fibrosis, or there may be a fatty replacement of the muscle fibres. An interfascicular fibrosis is also seen. The above changes result in a permanent loss of function or contractile power, and muscle atrophy may be apparent as early as fourteen days after the onset of the paralysis.

As described previously, the virus may not cause irreparable damage to the nerve cells and therefore the paresis or paralysis may not be permanent and there is then only a transient impairment of function. In the incomplete lesions it is found that some portions of the muscle function nor-

mally whilst others degenerate. In the presence of a permanent disintegration of a subtotal number of units in a muscle, an improvement in its strength occurs as a result of two factors. These are the reintroduction of muscular co-ordination and a hypertrophy of the remaining muscle fibres, and not from the new formation of muscle cells.

Some American workers stress the importance of a hypertonic condition of the affected muscles and report that it occurs in 100% of their cases in the acute phase. It appears that any muscle may be involved, but this phenomenon is discussed fully in the section on muscle spasm. This hypertonicity may cause permanent changes by fibrosis and contraction of the affected muscles.

The effects upon the muscle appear to be secondary to the nerve damage, but the most recent investigations tend to disprove this in some cases. It has been found that the Cocksackie virus can attack the muscle fibres directly.

Recently the condition of the heart muscle has been investigated. Interstitial myocarditis has been found frequently in fatal cases and it is present in direct proportion to the severity of the disease. The affected areas show inflammatory changes of different intensities, and a focal necrosis of the muscle fibres.

Our attention should now be directed to the bony skeleton, and it is found that the bones of the affected limb or limbs undergo atrophy and decalcification. Their shape may also be altered and this, coupled with the above changes, renders them less resistant to pressures and strains. These changes may be attributed to disuse of the limb, alterations in the local blood supply, or to primary neurotrophic disturbances.

In a child who has suffered from poliomyelitis, the affected limb may grow more slowly than its fellow, and the younger the victim the more noticeable is this effect, especially if the lower limb is the part involved. This factor, if coupled with the action of the unaffected muscles, may lead to a marked deformity.

The joints may also be affected, but this is usually the result of inadequate treatment or faulty supervision during treatment. If the appropriate treatment is not instituted early,

the capsule and ligaments may be subjected to severe strains. This will eventually lead to a marked weakness of the joint, as the result of the stretching of these structures. The joints will therefore become unduly mobile or flail, and subluxation or dislocation may occur.

Another deformity found frequently is pes cavus which is due to contracture of the plantar fascia.

General enlargement and congestion of the lymphatic tissues of the body are frequently seen. These are usually most marked in the region of the ileo-caecal junction and in the mesenteric lymph glands, and if Peyer's patches are examined they will be found to show hyperplasia. In a few cases they will be ulcerated and the lymph follicles filled with granular necrotic material. Proliferation of the reticular and endothelial tissues may also be seen.

If the patient dies in the acute stage, the following changes may be present in the abdominal organs. If the liver and kidneys are examined microscopically, they will show parenchymatous degeneration. The spleen may be enlarged, with hyperplastic follicles and prominent germinal centres, and degenerated and necrotic cells may be present.

In discussing the probable portals of entry of the virus, the alimentary tract was mentioned, and it is therefore fitting to point out the coincidence of poliomyelitis in patients who are suffering from dysentery or who have had a recent attack.

Cook *et al.* (1951) pointed out that the occurrence of gastro-intestinal ulceration and/or perforation with or without haemorrhage in cases of polioencephalitis is a much more common symptom than was formerly believed. They strongly advise that a routine guaiac test is carried out, so that any gastro-intestinal bleeding can be detected.

Finally a word on laboratory investigations apart from the routine chemical and cytological examination of the cerebrospinal fluid. If virus investigations are desired, the following specimens should be obtained and kept in a refrigerator until they are collected by the pathologist.

- (1) Five ccs. of serum from a definite or suspected case taken both in the acute and convalescent stages

- (2) A specimen of cerebrospinal fluid or of fresh post-mortem material from cases in which the meningeal or encephalitic signs and symptoms predominated.
- (3) Post-mortem specimens of brain or spinal cord, preserved in equal parts of glycerine and normal saline.

BIBLIOGRAPHY

58.
BRIDGE *et al* (1946) *Amer J Dis Child*, 72, 501.
COOK, C. D., *et al.* (1951) *Pediatrics*, 7, 415
FADER, H K., *et al* (1947) *Proc Soc exp. Biol Med. N.Y.*, 66, 103.
FISCHER, A E., and STILLERMAN, M. (1938) *J Amer. med. Ass.*, 110,
569.
Epilepsia 9 (1917) *Lancet* 88, 497.
J Amer. med Ass, 53, 1639.
J Amer med Ass, 53, 1913
J Amer med Ass., 54, 1780.
Bull Johns Hopk Hosp, 69,
HOWE, H A., and BODIAN, D (1942). *J Pediat*, 21, 713
JUNGBLUT, C W., and DALLDORF, G. (1943) *Amer. J. publ. Hlth*,
33, 169.
LANDSTEINER, K. (1908) *Sem méd. (Paris)*, 28, 620
LEVADITI, C., *et al.* (1936) *C R Acad Sci (Paris)*, 203, 899.
MAYR, O. (1932) *Z orthop Chir*, 57, 429.
NELSON, N (1946) *Science*, 104, 49.
NEUMANN, P. H. (1951) *Natl Bur Stand*, 56, 266
NEUSTAEDTER, M (1951) *Ann Surg*, 134, 65.
SHEINKER, I M (194)
STILL, G F. (1930)
TAYLOR, F. (1878)

CHAPTER III

PROPHYLAXIS

COMPULSORY notification should include abortive and non-paralytic cases as these play a major part in the spread of the infection, being much more prevalent than the frank hospital paralytic case.

All children who show any evidence of illness such as fever, nausea, vertigo, retention of urine, pain in the head, neck, back, limbs, chest or trunk, with perhaps some stiffness of the neck or spine should be isolated in bed for one week. They should have the maximum amount of fresh air and sunshine. Other contacts should be carefully observed, and at the first sign of illness they should be quarantined for two weeks.

The isolation period usually recommended is three weeks, but as 50% of patients are still excreting the virus in their stools after this time, it is obviously theoretically too short. Brown *et al* (1948) showed that 20% of the family contacts excrete the virus and therefore ideally isolation should include the whole family. To be effective, rigid quarantine should be enforced for three to eight weeks, i.e. until the virus can no longer be isolated from the stools. Not only is this rigid isolation impractical, but it is ineffective in preventing the spread of the disease because of the large number of sub-clinical cases, abortive cases and carriers who walk about undetected.

Flexner and Lewis (1910) demonstrated that the virus is neutralized by the serum of cases of human poliomyelitis, and by that of monkeys suffering from experimental poliomyelitis. In the same year, Bryant stated that epidemic poliomyelitis is a contagious and infectious disease which is amenable to local pharyngeal treatment.

Craster (1916) recommended that the following methods be adopted:

- (1) Isolation of the patient and attendant during the febrile period.

- (2) Quarantine of the family, except the wage earner.
- (3) Placarding.
- (4) Fumigation.
- (5) Hospitalization.
- (6) Prohibition of public gatherings of children.
- (7) Postponement of the opening of schools for three weeks
- (8) Permits and health certificates for children entering or leaving an affected area.

To ensure effective quarantine, Ball (1917) recommended the setting up of an efficient team consisting of a physician, a paediatrician and a neurologist, so that an accurate diagnosis could be made at the earliest possible date.

During epidemics, the infectivity of non-paralytic and preparalytic cases must be borne in mind, and early recognition of these, with isolation during the febrile period, is imperative.

1. Schools and Nurseries

Unless there are definite contra-indications, schools should not be closed because this will not control the infection and it may even tend to increase it. Children who are free to gather in groups in playing-grounds and streets are more likely to come into contact with adult carriers. All that is necessary is that a daily inspection should be made by the school medical officer. If a child is known to be an intimate contact, he should be kept away from school for three weeks from the date of exposure. Owing to the great susceptibility of very young children, nurseries should be closed if more than one case occurs in them.

2. Crowds

All persons should avoid crowds, and attendance at places where overcrowding can occur, e.g. theatres, cinemas and parties. It is interesting to note that in our series we encountered fairly frequently a history where all the members of the household suffered with "head colds."

3. Swimming-baths and Paddling-pools

As it has been found that the virus is destroyed by exposure to 0.2 PPM chloride for ten minutes, chlorinated swimming-baths need not be closed unless there is definite proof that a patient has become infected there. Frequent bacteriological examination will reveal whether the chlorination is effective, but as the strains of the virus vary in their resistance to chlorine, and especially if there is much organic matter present, the virus may not be killed by the usual amounts of chlorine used. Only a limited number of children should be admitted at one time, and then only for a restricted period, because exhausting exercise may predispose to the paralytic stage in an infected child.

Hired swimming-suits and towels should be washed in hot water and laundered before re-use and the floors of the changing rooms disinfected.

All fresh water paddling-pools should be drained, but river bathing is perfectly safe provided that no sewage pipe drains into the river.

4. Exercise

Strenuous and unusual forms of exercise must be avoided at all costs during the preparalytic stage, as this may determine the onset of a severe or even fatal paralysis. It is therefore reasonable to forbid cross-country runs, inter-house competitions, etc., as soon as a case is suspected. Fatigue must be avoided after minor injuries, operations and sunburn. Six of our cases gave a history of bathing and exercise rapidly followed by the onset of paralysis.

The relation between physical activity and the later development of paralysis in poliomyelitis is important both practically and theoretically. Ritchie Russell (1947) found that there was no correlation between physical activity in the three days before the onset and the degree of the subsequent paralysis, but he concluded that physical activity immediately after the onset predisposed to severe paralysis. Patients who rested in bed from the start of their prodromal symptoms rarely developed a disabling paralysis. These findings were confirmed by Hargreaves in 1948.

5. Personal Hygiene and Posture

Parents should insist on children washing their hands after the use of the toilet and before meals. Attention should be paid to the regular evacuation of the bowels, and the maintenance of a good posture is most important, even although there is no evidence at present that it is of specific importance.

6. Food

Personal hygiene must be of the highest order in all those handling food. Water of doubtful purity and unpasteurized milk should not be drunk, and fresh fruits and vegetables should be washed, peeled or scraped before consumption. Protective foods should be given in adequate amounts.

7. Flies and Vermin

Anti-fly measures and destruction of vermin should be intensified and the greatest care taken in the disposal of garbage, excreta and sewage. Food bins should have tightly fitting lids and be emptied regularly.

8. Operations and Injections

Cases occurred which seemed to be adversely affected by local injury, operation, injection or some other physical factor. Paralytic poliomyelitis usually developed four to twenty-one days after the local trauma. The paralysis was maximal or confined to that part of the body which was the site of the trauma. Several of our cases had a previous history of recent acute tonsillitis, tonsillectomy or dental extraction. Three had had a severe attack of measles seven to ten days previously, and in one family a brother and sister gave a history of recent measles. It is advisable that dental extractions, tonsillectomy and all elective operations on the ear, nose and throat or intestinal tract should be avoided during an epidemic. Mass inoculation against diphtheria and whooping-cough should be restricted to the non-epidemic periods of the year.

One of our cases had an attack of acute appendicitis one

month after admission and the operation was followed by an increased weakness of the right leg

A recent article in the *British Medical Journal* calls attention to the dangers of penicillin injections in children during epidemics of poliomyelitis. Six of ten paralysed children had had penicillin injections in the buttock or thigh and in all six paralysis was confined to the legs. Injections to children during poliomyelitis epidemics should therefore be kept to a minimum as it has been shown that oral penicillin usually gives satisfactory serum levels

9. Serum

Draper (1931) showed that although immune human serum can block paralysis in monkeys infected with the virus, it appears to be ineffective in humans, unless it is given in the brief interval between the moment of the choroid plexus penetration and the virus-cell union. Some observers advise the injection of 20 ccs. of convalescent serum or mixed adult serum during an epidemic, but it is quite obvious that if it is given at any stage after the infection, it must fail completely to influence the severity of the paralysis or the mortality rate.

In 1933 it was stated by Dornedden that the only prospect for prevention of paralysis and a fatal outcome is afforded by intravenous injection of convalescent serum at the onset of the disease.

Keller (1935) stated that in his opinion the application of convalescent serum after the onset of paralysis was useless and in fact harmful. However, he raised no objection to the transfusion of adult blood or blood from both parents.

In 1932 and 1935 Fairbrother advocated the prophylactic intramuscular injection of human convalescent serum, immune horse serum or tested serum of humans, and gargling with oxidizing substances, for all contacts in addition to the usual isolation.

Forrester-Brown (1939) pointed out that experiments prove that convalescent serum only protects the animal if it is injected at the same time as the virus, and naturally concludes that it is useless to give the serum after diagnosis.

Active immunization with the virus attenuated by ultra-violet irradiation appears to be effective in protecting mice, but it is not as yet applicable to man.

Passive protection against poliomyelitis may be given by means of gamma globulin but this is not the answer to the problem of securing long-term protection against the disease. The use of gamma globulin as a general protective measure is neither possible nor advisable and its use should be limited to carefully selected cases. Unfortunately absolute proof of its efficiency does not exist.

Active immunization produces prolonged immunity, but it must be safe, free from pathogenic properties due to the presence of foreign protein, and must contain all three types of the poliomyelitis virus.

The use of living attenuated viruses as immunizing agents would appear to be the best method for securing long-lasting and safe protection.

It has been known that antibody formation may result from the oral administration of certain strains of living virus in monkeys.

10. Deformities

Lastly, if poliomyelitis is suspected, a splint should be applied to protect and support the joints in the position of neutral muscle pull. The tissues should also be kept warm.

These precautions may help to prevent deformity and minimize disability.

BIBLIOGRAPHY

- BALL, C R (1917) *St Paul med J*, 19, 229
 BROWN, C G, *et al* (1948) *J. exp Med*, 87, 21
 BRYANT, W S (1910) *New York med J*, 92, 1215
 CRASTER, C V (1916) *Trans Amer Ass Study Prev. Inf. Mort.*, 7, 187
 DORNEDDEN, H (1933) *Gesund Fürs Kindesalter*, 8, 216
 DRAPER, G (1931) *J Amer med Assn*, 97, 1139
 FAIRBROTHER, R W (1932) *Clin J*, 61, 205
 ——— (1935) *New York med Ass*, 55, 662
 ——— (1936) *New York med Ass*, 55, 662

CHAPTER IV

CLINICAL FEATURES

It must first of all be stated that this disease requires the diagnosis to be made on purely clinical grounds because as yet the virus cannot be detected by any specific laboratory test. The attacks appear to be irrespective of previous health, social class or environment. The symptoms may be so mild that no definite diagnosis can be made, and yet the patient may be highly infective. Certain contacts can act as carriers and spread the infection, although they themselves appear to be perfectly healthy. Early and accurate diagnosis is therefore essential so that adequate measures can be taken to limit the spread of the disease, and to minimize the patient's suffering.

Numerous classifications of poliomyelitis have been attempted, but it is my intention to stress only the main clinical features which can be observed at the beginning of the illness and after the various regions of the nervous system are involved. It is important to note that only a few signs and symptoms may be present and that one type may overlap the other. Also the first symptom may be the onset of paralysis or mental confusion, without any other prodromal symptoms, and further, the disease may be arrested at any stage. To avoid missing the earliest signs, a systematic examination is necessary.

The severity of the signs and symptoms varies greatly in different epidemics, and there is a marked difference in the distribution and severity of the paralysis in each outbreak. During the 1947 epidemic in Great Britain, the distribution was as follows —

Paralysis of limbs and trunk	63%
Cranial nerves chiefly affected	10·3%
No paralysis present	23·3%

The severity of the paralysis appears to depend upon the type and virulence of the virus, the age of the patient and

the part of the nervous system involved. However, in the majority of cases the diagnosis can be made within twenty-four hours of the onset of the symptoms, especially during an epidemic. As a rule, after the onset of fever, the earliest paralysis to appear is that resulting from damage to the cranial nerves. It must not, however, be assumed that cranial nerve palsies are a very common occurrence as it is rare for more than one-third of the cases to be so affected.

Careful and repeated observations are necessary if complications are to be detected and treated promptly. In some cases of poliomyelitis, when the patient is very ill, it is extremely difficult and sometimes quite unjustifiable to make an accurate day to day detailed clinical examination.

There are without doubt a large number of abortive cases which are missed or remain undiagnosed and which because of this mild attack are later found to have a natural resistance to the virus.

ABORTIVE INFECTION

It is most important from the epidemiological point of view to remember that poliomyelitis may be represented in a particular area entirely by cases of the abortive type. Even during an epidemic the diagnosis may be only presumptive. In this period of the infection the poliomyelitis has not progressed beyond the systemic stage in a person who has been exposed to, or resides in the same house as a known case. There are no signs of inflammation of the central nervous system nor is there any permanent damage to health.

These abortive cases should never be admitted to hospital, because admission is justifiable only when neurological signs develop. For this reason, daily observation by the physician is necessary so that the earliest signs can be detected. There is no doubt that non-paralytic and often ambulant cases play a much greater part in the spread of poliomyelitis than the frankly paralytic patient in hospital.

The prodromal stage may last for a few hours to fourteen days and the symptoms are those which are common to certain other infections. In my series of cases, the time

between the onset of symptoms and that of notification was as follows:

TABLE IV

No of days	No of cases
0	22
1	41
2	43
3	67
4	45
5	42
6	39
7	33
8	27
9	24
10	22
11	15
12	14
12+	66
	—
	Total 500

In the initial stages, the symptoms are those of a general systemic disturbance and are very similar to those found in any infection of the respiratory tract by a virus. The patient may complain only of a slight head cold, general malaise, shivering or a mild upset of the gastro-intestinal tract.

At first the patient may be afebrile and feel quite well, but in 80% to 90% of cases the temperature is found to be moderately raised. Within twenty-four hours of the onset, it usually rises to 100°–103° F, but as this lasts only from three to six days, it can pass unnoticed. In my series, the feverish cases were as follows:

Age group 0–5 years	83%
Age group 5–15 years	92%
Age group 15+ years	77%

Rigors occurred in 23% of cases. The pulse is usually only slightly or moderately increased in rate whilst the respira-

tions remain unaltered in rate, rhythm or depth, but occasionally there may be pain in the chest.

The face is usually flushed, and there is a definite circumoral pallor or occasionally a mild diffuse erythema may be seen over the whole body. If a tourniquet is applied to a limb, fine petechiae may be produced in the skin. The patient may also suffer from profuse sweating. Epistaxis was recorded in one of our cases aged 11 years.

In 20% of cases the patient complains of a sore throat which on examination may show a varying degree of redness, but oedema and inflammatory exudates are rarely seen. Sore throat was complained of by 28% of our cases, but Pohl (1947) gave a figure of 10% in his review. 19% of our cases had catarrh of the nose and 16% had catarrh of the chest. The complaint of sore throat occurs ten times more frequently in cases in which bulbar symptoms ultimately develop than in those which eventually show paralysis of spinal origin. Bulbar paralysis must therefore be watched for in these cases.

Headache is usually one of the first symptoms. It occurs in 60% to 80% of cases within twenty-four hours and may be frontal, vertical, occipital, temporal or generalized. Between 50% and 92% of the patients complained of headache in the 0-5 and 5-15 age groups, whilst the main distribution in this series was 45% for frontal headache in the 0-5 age group and 92.6% for the 5-15 age group. Occipital headache varied from 15% to 25% in the 0-5 and over 15 age groups respectively. If the headache is occipital, it may be due to congestion of the cerebral vessels, or to pain associated with nuchal rigidity. Although moderately severe, the headache is rarely intense, but it may be persistent or throbbing in character. Clinically it is very difficult to be sure of this symptom in infants.

The tongue is usually furred and nausea and anorexia are frequently present, giving rise to marked distress. Loss of appetite was present in 72% of our cases.

Vomiting occurs early in 60% to 80% of cases and is usually of sudden onset, repeated and forcible, but lasts only for approximately forty-eight hours. In our series, vomiting

varied from 42% to 63% of cases in the 0-5, 5-15 and 15+ age groups

Diarrhoea may also be present in this stage and was found in 10% of our cases

General muscular aching and lethargy, or restlessness and fretfulness may be present, but these symptoms are more commonly found in the preparalytic stage

In this stage the changes in the cerebrospinal fluid are not specific but only suggestive

If the disease does not progress any further, it is usually found that the patient remains lethargic for several days

PREPARALYTIC STAGE

In the preparalytic stage there is practically no difference in its symptomatology and that of the current infectious diseases, but it is comparatively easy to make the diagnosis during an epidemic if nuchal rigidity or stiffness of the spine are also present. One pitfall is that a large number of cases suspected of being non-paralytic poliomyelitis, may be due to other viral infections

A history of exposure to infection is usually but not invariably obtained, but the abortive stage may be so short that it is impossible to distinguish between it and the preparalytic stage which lasts only from one to three days. On the other hand, there may be an interval of two to three days during which time the patient feels remarkably well. It is most important to determine the onset of the signs of involvement of the central nervous system as compared with the onset of the initial febrile attack, this being usually thirty-six hours

This stage is characterized by all the signs and symptoms of the abortive type along with those of involvement of the nervous system but without any evidence of paresis or paralysis. The severity of the spinal and meningeal symptoms, however, varies greatly from case to case

In a few cases after the initial rise of temperature observed during the abortive stage, there may be a secondary rise. This secondary febrile phase is usually the first to be observed

by the physician, but its true significance may be missed owing to it being mistaken for the primary one. The temperature usually subsides in three to four days by lysis, rarely by crisis, in both the paralytic and non-paralytic types, but it may remain elevated for ten days. When the patient becomes afebrile, the signs of meningeal irritation disappear and there is only a minimal risk of further progress of the disease.

The pulse may be rapid or slow and this may be due to an early bulbar involvement. More recently it has been suggested that tachycardia is due to the direct action of the virus on the myocardium. An alteration in the heart muscle can be shown histologically or by electrocardiographic tracings.

The general symptoms in the preparalytic stage may be slight or severe, but in themselves they are not diagnostic. Mental irritability may be one of the earliest symptoms and it is usually followed by drowsiness which may be very marked in some cases, or occasionally it may alternate with periods of intense irritability. In a few cases, however, the excitability persists and may even be increased. In other cases the patient may be nervous, fretful, restless, apprehensive and anxious-looking, or he may be lethargic, apathetic, prostrated or stuporose. Rarely the patient may be critically ill, comatose or suffering from convulsions. 2% of our series were markedly comatose and 3% had generalized convulsions. The patient may lie quietly in bed, resenting being disturbed. Definite signs of meningeal irritation may be present, the headache being intense and vomiting a prominent symptom. Occasionally the sleep rhythm is inverted, the patient being very restless during the night and sleepy during the day. 41% of our patients suffered from insomnia and 5% had marked stupor. One of our patients, a girl of 23 years, suffered from marked emotional upset. It was noticeable that when convulsions occurred, it was usually in the younger patients, whilst delirium was noticed in patients over 30 years of age.

Slight enlargement and tenderness of the cervical glands is an important sign and should be sought for in all cases.

Conjunctival congestion and glazed cornea have been noticed in early cases, and rapid jerking nystagmoid movements of varying amplitude usually affecting both eyes. These movements are present in the horizontal, vertical, and oblique planes, and occasionally there is a rotatory movement. They occur in decrescendo, are intensified by motion but do not interfere with vision. These signs have been attributed to a lesion of the basal ganglia with or without a disturbance of the cerebellum or its connections. Diplopia and blurred vision have also been observed about the fourth day. In the preparalytic type, as in the abortive, the eye symptoms usually disappear spontaneously during convalescence.

Gastro-intestinal atony, due to sympathetic involvement, is frequently present and gives rise to obstinate constipation and abdominal distension, but in approximately 10% of cases, diarrhoea is present with very offensive stools. Constipation may also be caused by inactivity, weakness, paralysis of the abdominal muscles, loss of habit or fear of pain. In our series, constipation was present in 56% of cases.

The number of patients who complain of bladder symptoms varies greatly, and figures between 2% and 65% have been quoted. An important point is that these symptoms, including dysuria, may be noted some time before the onset of the paralysis of the limbs. The patient may complain early of difficulty in starting micturition or there may be a complete retention of urine with an accompanying overflow incontinence. The paralysis is transitory and recovery is usually complete within a week. Retention of urine was present in 8% of our cases. A urinary output chart should be kept and other disturbances of the bladder watched for.

True incontinence may occur from the onset but it invariably recovers, although it may take several weeks to do so. If infants are affected, the involvement of the bladder may manifest itself by the persistence of enuresis for a number of years. Incontinence occurred in 6% of our cases.

Wright (1936) attributed the immediate urinary dysfunction to a peripheral neuritis involving the innervation of the bladder, with resulting painful and irritable bladder, spasmodic sphincters and acute retention. It has also been said

to be due to inflammatory oedema around the damaged anterior grey columns of the lumbar and dorsal region, interrupting the tracts linking the bladder centre in the cord with that in the hind-brain. Another theory is that it is produced by the involvement of the secondary centres in the tegmentum of the pons and the reticular substance of the medulla.

Toomey *et al.* (1943 *a* and *b*) attempted unsuccessfully to isolate the virus from the urine of patients with paralysis of the bladder, and also failed to produce paralysis of the bladder by injecting the virus into the bladders of rabbits.

The remote urinary complications are due to stasis, residual urine and infection from catheterization.

The superficial reflexes, e.g. the abdominal and cremasteric, are diminished or lost early. The tendon and skin reflexes may be exaggerated in the preparalytic febrile stage or they may be lost early and be asymmetrical.

It has been said that general hyperaesthesia is a constant finding in the early stages, although it varies greatly in its intensity. Other observers whilst agreeing that hyperaesthesia can occur either generally or locally, declare that it is seldom seen. Severe sacral pain may, however, be present in patients who often later develop a severe lower limb paralysis. The patient may be seen to lie in a peculiar position in an attempt to ease the pain which is increased by pressure on, or stretching of the muscles. Any attempt to persuade him to assume a reasonable posture either passively or by his own efforts, is markedly resented.

In the preparalytic stage, the sensory disturbances are said to be due to the early involvement of the posterior root ganglia. Pain and tenderness in paralysed cases can nevertheless be present in non-paralysed muscles, because the inflammation of the sensory neurones does not necessarily occur at the same level as the anterior horn cell involvement.

Hyperirritability as demonstrated by local pain, paraesthesia and stiffness of the neck and back muscles, is usually attributed to involvement of the nerve roots, internuncial neurones, posterior root ganglia or their surrounding meninges. Areas of hypoaesthesia and anaesthesia are rare.

Frohring *et al* (1945) measured the changes in the vibratory sense of patients with poliomyelitis by means of the pallesthesiometer. They demonstrated that both the sensory and internuncial neurones were affected

Changes in the cerebrospinal fluid may also be seen in this stage

It will be obvious that a definite diagnosis cannot always

be made at this stage. The patient must be watched carefully for another two weeks in order to exclude poliomyelitis definitely

PARALYTIC STAGE

In the 1947 epidemic, of every one hundred persons who showed clinical signs and symptoms of infection, less than five developed into a frank paralytic case. The clinical features vary greatly and any of the abortive and preparalytic symptoms may have been present in a patient who has now developed paresis or paralysis. It is important to note that the symptoms in the preparalytic stage give no indication of the degree or distribution of the ensuing paralysis. Pohl (1947) stated that 34.6% of his cases showed no paralysis at any time, whilst 65.4% of cases progressed to paralysis.

The final diagnosis of poliomyelitis appears to depend mainly on the number of abortive and preparalytic cases which occur in the epidemic under review. In the epidemics in Malta and Mauritius, the majority of cases were paralytic. Nevertheless to delay in making a definite diagnosis until the paralysis has set in is quite unjustifiable, because valuable time is lost before the initiation of the appropriate treatment.

Walton (1907) stated that no extension of the paralysis was to be expected after the fever had subsided. In the so-called "dromedary" type of temperature chart, the febrile illness usually lasts from one to three days to be followed

by a period of one to four days in which the patient feels remarkably well. This, however, is followed by a secondary rise in temperature which lasts for one to three days, and is usually accompanied by a spread of the paralysis. Another rare type is that seen at the end of two or three weeks of apparent inactivity, and it is manifested by an increased pain and tenderness of the muscles and a marked spread of the paralysis.

If the temperature remains elevated, the paresis may progress but to a less extent on each subsequent day. In other cases, each extension of the paralysis may be associated with a temporary rise of temperature. The degree of elevation of the temperature does not appear to be of any diagnostic significance, although a prolonged fever appears to affect the prognosis adversely.

Nissen (1947) stated that the tongue was moist and of a cold bluish colour. In all cases with paralysis, he noted that there were small macules similar to flea bites around the margin, and that these changes lasted for approximately two weeks. I have been unable to verify this, although several of our cases had furred tongues.

Anoxia may be due to abductor paralysis of the vocal cords, or from obstruction of the airways by pools of mucus, saliva or vomitus. Other causes may be a reflex closure of the glottis, and pulmonary oedema with a consequent decrease in the alveolar absorptive surface. If there is a diminished cough reflex, aspiration may be required to remove obstructive fluids from the lungs. Anoxia produces damage to the nerve cells, and overaction of the weakened respiratory muscles increases fatigue and may actually increase the paralysis.

Ager (1907) believed that the earliest manifestation of the disease was a gastro-intestinal disturbance accompanied by fever, and that paralysis appeared one to two weeks later. Occasionally the patient may be incontinent of faeces from paralysis of the rectal sphincters, and unfortunately he may remain so permanently.

Paralysis of the bladder is frequently associated with paralysis of the lower abdominal and thigh muscles. In 40%

of cases in one series in which both legs alone were severely paralysed, there was a retention of urine for two to three days, normal control being then spontaneously re-established. In the spinal form of paralysis, the bladder symptoms and dilatation of the ureters with a secondary pyelonephritis, have been attributed to an involvement of the sympathetic nervous system.

As a rule, the paralysis appears about the second to the fifth day after the onset of fever, or one to two days after the onset of the meningeal symptoms. As observed by Russell in 1947, the meningitic symptoms usually abate and the patient feels better before the paralysis develops. On the other hand, the paralysis may appear extremely quickly without any warning especially in children in the 0-5 age group. The paralysis may, however, be delayed for several days, but in over 95% of cases the maximum degree of paralysis is seen between the first and fifteenth days of the onset. In a small percentage of cases the paralysis may only become obvious after the second week. In our series of cases the time which elapsed between the onset of the disease and the first appearance of paralysis was as follows

TABLE V

No of days	No of cases	Percentage
1	30	16.8
2	34	19.2
3	39	22
4	24	13.5
5	12	6.7
6	7	3.9
7	14	7.9
7-14	11	6.1
14-21	5	2.8
21-28	2	1.1

Paralysis may be maximum in its severity and distribution at the onset, or it may, during epidemics, progress for one to three days especially in adolescents and adults. Walton

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may at first be stimulated. recorded, 20 were normal, 20 were diminished and 12 absent. In no case were they exaggerated. It should be noted that there can be a complete loss of the knee jerks and yet there is no detectable paralysis.

In some cases with severely paralysed lower limbs, the intensity of the inflammation in the affected region of the cord leads to a spreading oedema into the white matter. This in turn leads to a transitory extensor plantar response in one or both limbs.

In the later stages, poor circulation and trophic changes result in local sensory disturbances.

Smith *et al* (1949) showed that there is clinical evidence of the involvement of the sympathetic nervous system, which may manifest itself in many regions.

Cervical region.	Horner's syndrome which is manifested in unilateral cases by myosis, drooping of the upper eyelid and sweating of one side of the face
Thoracic region	Spasm of the pulmonary blood-vessels with dilatation of the right side of the heart.
Gastric region.	Pylorospasm
Intestinal region.	Constipation or diarrhoea
Rectal region.	Intestinal obstruction
Bladder region.	Retention or incontinence of urine.
Skin.	Angioparesis or angiospasm with fall in skin temperature
Blood-vessels.	Spasm in paralytic and non-paralytic limbs. Chilblains of intractable type

The general systemic symptoms usually subside within four days, and the disease progresses no further, but the virus may be excreted for several days or weeks, and therefore there is danger of spread from the nasopharyngeal secretions or faeces during that time.

Before leaving this section, it is necessary to draw attention

(1907) observed that the onset of paralysis was more retarded in adults than in infants, and that the extension from one group of muscles to another was less rapid. One hundred and nineteen of our cases, i.e. 70%, showed a sudden onset of paralysis, and 51 cases, i.e. 30%, showed a gradual one. The paralysis was increased within twenty-four hours in 79 cases (55%), was stationary in 45 cases (31%), and decreased in 20 cases (14%). In 45% of cases the paralysis was of an ascending type and was descending in 55%. Frequent thorough muscle testing may show a spread of the paralysis for several days after the onset, and certain observers have stated that the electromyographic examinations have shown that there is a definite spread after the febrile phase has passed. It is therefore essential in the early stages to give a very guarded prognosis.

During an epidemic, well over 50% of infected patients will recover fully without showing any signs of paralysis.

Loss of sensation can usually be detected if looked for clinically; for example, a loss of pain and temperature sense may be detected in the first week of the illness. It can be bilateral and occur on the opposite side below the lesion. The patient complains of pain in the feet during the night but not during the day. He may also complain of severe girdle pain and in one of our cases this feeling of constriction around the waist was very marked for ten days before the onset of paralysis of both lower limbs and abdominal muscles. Another case complained of pain and weakness in the legs for twelve hours before the onset of the paralysis, whilst another complained of numbness of both legs.

In the age group 0-5 years the loss of sensation appeared to be widely distributed, e.g. the sole of the foot, the lateral aspect of both legs, the plantar aspect of the foot, etc. These symptoms are usually only temporary and may last for two to five days.

The tendon reflexes diminish rapidly after the onset of the paralysis and finally disappear, or they may appear to be normal at the onset, and in a few hours the paralysis may develop. It is important to note that the character of the reflexes can change from one examination to another. Where

immunity. Modern sanitation also reduces the risk of sub-clinical infection from alimentary sources in childhood. The mid-brain, pons and medulla and any cranial nerve nuclei or tracts, situated in, or passing through this part of the brain, may be involved.

The preparalytic stage is usually shorter than in the purely spinal type lasting one to two days, and it may be preceded by high fever, rapid pulse, severe headache and nuchal rigidity. The fever may last for ten days and it can be prolonged even longer if secondary respiratory infection occurs. Severe, forceful and unremitting vomiting may also be present along with a sore throat and laryngitis. The patient may show evidence of extreme malaise and drowsiness, or intense excitement, marked restlessness, irritability and apprehension may be the chief signs.

As Boines (1947) pointed out, an early diagnosis of polio-encephalitis can be made if the muscles supplied by the cranial nerves are carefully examined. The first indication of paralysis may be dysphagia or a slurring of speech. Weakness or loss of power of coughing or an ineffective cough may also be present, and secretions may collect in the mouth and air passages.

Wolfe (1894) reported on a case with involvement of the first, third, fourth, sixth, seventh and ninth cranial nerves, and Steiglitz (1897) described a case in which the facial palsy preceded the constitutional symptoms and consequent paralysis by five days. In our series, thirty cases had various combinations of involvement of the cranial nerves of which the following were the most common. Seven cases had involvement of the ninth cranial nerve, and in three cases the seventh and ninth were both involved. Other combinations noted were the third and tenth, fifth and seventh, seventh and twelfth, ninth, tenth and eleventh, and in one case the ninth, tenth, eleventh and twelfth.

Third, Fourth and Sixth Cranial Nerves

As the result of involvement of these nerves, diplopia, strabismus and unilateral or bilateral ptosis may occur. The

once more to the important fact that has now been fairly well established, namely, the performance of any exercise after the onset of the febrile illness is extremely dangerous, and may leave the patient extensively and permanently paralysed. Even the restlessness exhibited by some patients may be harmful.

MYELITIC FORM OF POLIOMYELITIS

A common form is characterized by an upper and lower motor neurone lesion accompanied by a temporary paraesthesia.

There is flaccid paraplegia with extensor plantar responses. Retention of urine is also found. There is a sensory loss over the abdomen and residual spastic paraplegia.

NEURITIC FORM OF POLIOMYELITIS

The patient complains of severe pain, worse on movement, in the affected limb. There is tenderness on pressure of the nerves and muscles. There may, however, be no paralysis but if there is, it is followed by wasting. The tendon reflexes are lost and there may be disturbances of sensation.

POLIOENCEPHALITIS

BULBAR FORM OF POLIOMYELITIS

It is significant to note that the intercerebral inoculation of the virus of poliomyelitis into monkeys invariably produces the spinal type of poliomyelitis. It is also noted that adults are much more likely to develop this type of poliomyelitis than infants, and that the majority of deaths occur in this group.

of the bulbar type. There is no doubt that there is an increase in the proportion of cases of polioencephalitis. The accepted explanation appears to be the larger number of patients which reach adolescent and adult life without developing

immunity. Modern sanitation also reduces the risk of sub-clinical infection from alimentary sources in childhood. The mid-brain, pons and medulla and any cranial nerve nuclei or tracts, situated in, or passing through this part of the brain, may be involved.

The preparalytic stage is usually shorter than in the purely spinal type lasting one to two days, and it may be preceded by high fever, rapid pulse, severe headache and nuchal rigidity. The fever may last for ten days and it can be prolonged even longer if secondary respiratory infection occurs. Severe, forceful and unremitting vomiting may also be present along with a sore throat and laryngitis. The patient may show evidence of extreme malaise and drowsiness, or intense excitement, marked restlessness, irritability and apprehension may be the chief signs.

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Third, Fourth and Sixth Cranial Nerves

As the result of involvement of these nerves, diplopia, strabismus and unilateral or bilateral ptosis may occur. The

patient may also suffer from photophobia, loss of accommodation, pupillary paralysis and occasionally nystagmus. In our series we observed unequal pupils in 2% of patients, diplopia in 6%, nystagmus in 4% and photophobia in 19%. One case suffered from intermittent blindness extending over a period of three days.

As each of these three cranial nerves may be involved individually, a brief description will be given:

1. *Oculomotor nerve.* A lesion of this nerve produces ptosis of the upper eyelid and an inability to open the eye. This is due to paralysis of the levator palpebrae superioris. The pupil is fixed in dilatation and the eyeball is turned outwards and downwards, due to paralysis of the superior, inferior and medial recti, and inferior oblique muscles.

2. *Trochlear nerve.* A lesion of this nerve produces paralysis of the superior oblique muscle, and when the patient looks downwards and outwards he has diplopia. In one of our cases there was involvement of the fourth cranial nerve which was the only sign of paralysis.

3. *Abducens nerve.* A lesion of this nerve produces paralysis of the external rectus muscle, the eyeball being turned inwards, and the patient unable to move it outwards beyond the mid-point.

Paralysis of conjugate deviation, due to damage to the intermediary centres, has occasionally been seen.

A most peculiar and relatively rare eye condition known as opsoclonia may also be observed. It is associated with a lesion of the basal ganglia, with or without a disturbance of the cerebellum or its connections in the brain-stem. If this phenomenon is present, it will be seen as a series of rapid and unequal ocular movements which may be horizontal, vertical or rotatory. They are worse at the beginning of the attack but tend to disappear as the gaze becomes fixed. These movements occur in groups of approximately six in number, the first being the greatest in amplitude and then occurring in decrescendo. They are intensified by emotion, but the

patient's vision is normal. They may also be accompanied by tremors of the lids, forehead, lips and fingers.

It has also been observed that the majority of patients treated in a mechanical respirator show signs of stasis of the retinal vessels and the optic papillae.

Fifth Cranial Nerve

A lesion of the motor root of the mandibular division of the trigeminal nerve results in a paresis of the muscles of mastication (except the buccinators) on that side. The patient is unable to bite firmly or to protrude the lower jaw. If both external and internal pterygoid muscles are involved, the side to side movement of the mandible is impaired.

Dechaume and Tardieu (1931) described a case in which atrophy of the left masseter was the only sequel. Williams (1947) reported on a case of a boy aged 6½ years, who, eighteen months after an attack of polioencephalitis had no demonstrable contraction of the temporal, masseter or external pterygoid muscles. Here the lesion appeared to be limited to both the trigeminal motor nuclei.

A bilateral paralysis of the muscles of mastication results in a dropping of the jaw, and the mouth remains open.

Seventh Cranial Nerve

A lesion of this nerve in polioencephalitis usually results in a total paralysis of the muscles of the lower part of the face on the opposite side, and a partial paralysis of the upper part. The movements of expression are lost, and the patient is unable effectively to smile, whistle or frown.

Steigman and Sabin (1949) reported on a seven-month-old infant where the virus was isolated from the bowel, and the antibody to the homologous strain of the virus appeared in convalescence. In this case the only paralysis was a unilateral one of the facial nerve.

deLavergne *et al* (1932) reported on the records of over 80 cases of isolated facial nerve paralysis and Kelleher (1947) found 5 cases during the London epidemic in 1947. Young (1945) stated that in his cases the facial nerve

paralysis was always confined to the left side. In our series there were 15 cases, one of which was bilateral.

Facial nerve paralysis is seldom seen as the only sign of paralysis, and it is usually accompanied by an involvement of the palate.

If complete recovery from the facial paralysis does not occur within one year, then there will be little or no further improvement.

Muscles Supplied by the Seventh Cranial Nerve

MUSCLES	MOVEMENT
Frontalis	Raises the eyebrows
Orbicularis oculi	Shuts the eyes.
Corrugator supercilii	Allows frowning
Procerus	Wrinkles the side of the nose.
Levator labii superioris	Widens the nasal opening.
Risorius	Allows smiling.
Levator anguli oris	Shows the upper molars
Triangularis	Shows the lower premolars.
Mentalis	Wrinkles the chin.
Buccinator	Allows blowing or whistling.
Orbicularis oris	Purses the lips.

Eighth Cranial Nerve

An involvement of the vestibular division of the auditory nerve with a disturbance of vestibular function is sometimes encountered, but it may be missed as it is usually of very brief duration.

Deafness has also been reported as due to involvement of the auditory nerve

Tenth Cranial Nerve

Involvement of the vagus nerve or the nucleus ambiguus leads to a unilateral or bilateral paresis or paralysis of the soft palate, pharynx and vocal cords. If the voice is nasal, paralysis of the soft palate should be suspected.

In unilateral laryngeal motor paralysis there is hoarseness

and stridor, and if there is bilateral abductor paralysis, there is complete aphonia, severe dyspnoea and stridor.

If both vagi are involved, there is tachycardia, and slowness and irregularity of breathing.

Brahdy and Lenarsky (1934) reported that approximately 65% of their cases with bulbar involvement complained of dysphagia, and Miller (1939) recorded a case in which the function of deglutition was the first to be paralysed and the last to be restored after eighty-six days of complete paralysis. This symptom varies greatly in intensity from simple regurgitation through the nose to a complete inability to swallow. In the latter case, it is due to paralysis of the pharyngeal constrictors. This inability to swallow, coupled with a difficulty in coughing, results in the pooling of saliva, mucus or vomitus in the throat. This causes obstruction to the airway and a temporary asphyxia, or later on an aspiration pneumonia.

Eleventh Cranial Nerve

A lesion of the spinal accessory nerve produces paralysis of the sternomastoid muscle, and the extent of the paralysis of the trapezius depends upon how much of it is supplied by the third and fourth cervical nerves. The curve of the neck is flattened and the scapula is displaced downwards and its vertical border displaced outwards.

If there is unilateral paralysis of the sternomastoid, there is weakness in drawing the head towards the shoulder of the same side and in rotating the head to look towards the opposite shoulder. In bilateral cases there is weakness in flexing the head and the cervical part of the vertebral column.

Twelfth Cranial Nerve

Hypoglossal nerve involvement results in paresis or paralysis of the intrinsic and extrinsic muscles of the corresponding half of the tongue which becomes wrinkled and atrophic. The patient has difficulty in the enunciation of consonants, syllables or words. When the tongue is protruded, it deviates towards the paralysed side.

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In unilateral laryngeal motor paralysis there is hoarseness

Lethargy and coma may precede death, although Brewis and Newbauer (1948) described a case which recovered after coma, fits, cerebellar signs and spastic weakness.

Certain investigators including Brown *et al.* (1948) conclude that the cerebral symptoms are due mainly to anoxaemia even although focal cerebral lesions have been found.

CEREBELLAR FORM OF POLIOMYELITIS

The patient complains of intense headache, vertigo and vomiting on moving the head. Nystagmus may also be present.

The ataxia in poliomyelitis is generally regarded as cerebellar in origin, and if so it is usually accompanied by retraction of the head and nuchal rigidity. This ataxia may, however, be cerebral or spinal in origin. A female patient aged 22 years showed all the signs of acute cerebellar ataxia, but did not develop any paralysis. The signs persisted in varying intensity for eleven days before the onset of the illness. Acute cerebellar ataxia occurred in 2% of our cases and vertigo in 18%.

Very rarely, there is also emotional instability, gross ocular tremor, and tremor of the head and limbs.

ANOMALOUS FORMS OF POLIOMYELITIS

In rare cases, slowly developing primary optic atrophy has been observed, and the muscles of respiration may alone be involved.

In 1903 Bonfa reported on a case with right facial paralysis, and deLavergne *et al.* (1932) pointed out that facial paralysis may be the only manifestation of the disease or that it may be accompanied by bulbar or spinal cord involvement.

It is seldom, however, that the disease is limited exclusively to one of the above types.

SUMMARY

(1) The incubation period from the time of exposure lasts for seven to fourteen days.

Since the nuclei of the glossopharyngeal, vagus, accessory and hypoglossal nerves are situated in the medulla in close proximity to each other, and if the clinical signs mentioned above are present, then the diagnosis of polioencephalitis should be quite clear. It should, however, be remembered that these nuclei may also be affected in other neuritic and encephalitic conditions.

CEREBRAL FORM OF POLIOMYELITIS

In this type the frontal, parietal and occipital areas of the cerebrum are involved.

According to Powell (1937) the most important points are the signs volunteered by the patient or his relatives, such as nuchal rigidity, drowsiness, etc., which are not present when the patient first comes under the observation of the doctor.

In the less severe cases there is fever with flushing of the face, vomiting, general malaise and listlessness. This may be accompanied by troublesome yawning, diplopia and insomnia or somnolence. In the more severe cases there is an extremely rapid tremor and twitching of the facial muscles and of the extremities. In the most severe cases the patient may become drowsy or even comatose, whilst in others there may be unilateral or bilateral convulsions extending over several hours. This may be followed by complete recovery, hemiplegia or paralysis of one limb with a subsequent retardation in the growth of that limb. Mental deterioration may occur, or athetosis, choreiform movements or epilepsy may be the sequelae.

In adults the mental symptoms may be very marked, and the patient may be irrational and suffer from hallucinations, marked confusion and anxiety. Other cases may be apprehensive, fretful and distressed out of all proportion to their pain or discomfort. Other symptoms which may be present are hyperexcitability, restlessness, irritability and delirium.

Forrester-Brown (1939) stated that the commonest cerebral changes are emotional instability with subtle changes in personality, and in adults a loss of the power of mental concentration, although recovery is usually complete.

BIBLIOGRAPHY

- AGER, L. C. (1907) *Long Is med J.*, 1, 491
 BOINES, G. J. (1947) *Delaware St med J.*, 19, 209
 BONFA, A. (1903) *S. Afr. med Rec.*, 1, 67.
 BRAHDY, M. III, and LENARSKY, M. (1934). *J. Amer med Ass.*, 103, 229
 BREWIS, H. G., and NEWBAUER, C. (1948) *Brit med J.*, 2, 416.
 BROWN, C. G., *et al* (1948) *J exp Med.*, 87, 21
 DECHAUNE, M., and TARDIEU, A. (1931) *Rev Stomatol, Paris*, 33, 223
 FORRESTER-BROWN, M. (1939) *Practitioner*, 142, 495
 FROTHINGHAM, W. C. *et al* (1945) *Am J Pathol*, 51, 69-90
 .
 .
 .
 POHL, J. F. (1947) *J Amer med Ass.*, 134, 1059
 POWELL, M. L. (1937) *Med J Aust.*, 24, 419
 SMITH, E., *et al* (1949) *J. Pediat.*, 34, 1
 STEIGMAN, A. J., and SABIN, A. B. (1949) *J exp. Med.*, 90, 349
 STIEGLITZ, L. (1897) *J nerv ment Dis.*, 24, 98
 TOOMEY, J. A., *et al* (1943a) *J Pediat.*, 23, 172
 TOOMEY, J. A., *et al* (1943b) *J Pediat.*, 23, 719
 WALTON, G. L. (1907) *Boston med surg J.*, 157, 719
 WILLIAMS, D. (1947) *Proc roy Soc Med.*, 40, 555
 WOLFE, S. (1894) *J nerv. ment Dis.*, 21, 229
 WRIGHT, B. W. (1936) *J Urol (Baltimore)*, 35, 618
 YOUNG, J. G. (1945) *Tex St J Med.*, 40, 527

(2) The systemic phase lasts for one to fourteen days and the following clinical features may be observed:

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|--------------------------------------|--|
| (1) Sick, fretful, restless patient. | (8) Vomiting. |
| (2) Moderate fever. | (9) Intestinal upset. |
| (3) Malaise. | (10) Enlargement of the cervical glands. |
| (4) Head cold. | (11) Occasional pain in the chest and limbs. |
| (5) Sore throat. | (12) Weakness. |
| (6) Injected pharynx. | |
| (7) Nausea. | |

95% of cases make a complete recovery whilst 1% to 5% progress to the preparalytic stage in one to four days

(3) The preparalytic stage lasts for seven to fourteen days, but may be as long as three weeks. The patient has an expression of impending disaster and is most apprehensive. He looks acutely ill, has a temperature of 102°–104° F., and may be completely prostrated. The face is flushed and the tongue furred. There is severe headache and backache and there may also be an increasing stiff neck and/or back. The muscles may be sore or tender, and there is pain in the neck and spine when bending the head forward. Tremor and ataxia may also be present. The reflexes are altered and may be exaggerated and asymmetrical. Occasionally there is vomiting, drowsiness, irritability, coma and nystagmus. Abnormalities are detected in the cerebrospinal fluid. The cells are usually increased in number up to 250/cmm. and are chiefly lymphocytes. The globulin test is positive and the glucose content may be normal or increased.

(4) The paralytic stage usually lasts for thirteen to eighteen days, but may be as long as five weeks. 50% of cases recover without signs of paralysis, but 10% to 50% are found to have some degree of paralysis. In this stage there is an early acute period when the lesions are active and there is muscle paresis or a flaccid paralysis. The reflexes are usually diminished or absent.

BIBLIOGRAPHY

- AGER, L. C. (1907) *Long Is med J*, 1, 491.
 BOINES, G. J. (1947) *Delaware St med J*, 19, 209.
 BONFA, A. (1903) *S Afr med Rec*, 1, 67.
 BRAHDY, M. B., and LENARSKY, M. (1934) *J Amer. med Ass.*, 103, 229.
 DICKSON, F. C., and NEWMAN, C. (1938) *Brit med J* 2 416.

, 33, 223.

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CHAPTER V

RESPIRATORY FAILURE

It is most important to recognize the difference between respiratory failure due to involvement of the respiratory centre and affection of the cervical and thoracic cord with paresis or paralysis of the primary muscles of respiration. It is not always possible to differentiate between the different types of respiratory failure and occasionally they may both be present in the same patient. Paralysis of the respiratory centre was found in only 4% of our cases and was usually confined to the 5-15 and 15+ age groups.

(1) INVOLVEMENT OF THE RESPIRATORY CENTRE

This may be preceded by a rise of temperature and pulse rate, cyanosis, sweating, anxiousness and apprehension. Periods of mental confusion and hiccup are often present.

Involvement of the respiratory centre is evidenced by an irregular jerky type of respiration which is completely without rhythm, or alternatively a sudden respiratory failure may occur. The respirations are shallow in depth and the intervals between them become more and more prolonged until the breathing stops completely. Pulmonary oedema occurs and Cheyne-Stokes' respirations usually precede the fatal termination. The patient dies suddenly of respiratory failure usually within twenty-four hours of admission to hospital. The above symptoms occur in spite of the fact that the intercostal muscles and diaphragm are normal and remain unparalysed.

Respiratory obstruction may be due to one or more of the following causes—

- (a) Extensive involvement of the lower cranial nerves with mechanical difficulty in swallowing and a tendency to asphyxia from the accumulation of mucus and saliva in the pharynx.
- (b) Abductor paralysis of the vocal cords.

- (c) Spasm of the larynx requiring immediate tracheotomy.
- (d) Pulmonary oedema
- (e) Atelectasis

Other factors which cause impairment of pulmonary ventilation are anaemia, abdominal distension and the use of sedatives. Hysteria must also be borne in mind.

Involvement of the respiratory centre and the cranial nerve nuclei is usually accompanied by involvement of the circulatory centre, and it can therefore very conveniently be discussed here.

In the early stages the face becomes markedly flushed and the lips bright red. The pulse rate, which may be regular or irregular, increases to 150 or more per minute, and is thready in character. Tachycardia and irregularity of the heart have been recorded and also hypertension, especially in children. In most cases, however, the blood-pressure falls, the skin becomes cold and clammy, and the patient is apprehensive, restless and confused.

As Guyton (1948) pointed out, most of the symptoms are possibly explained by the large number of lesions in the brain stem, but because of its marked reparative powers these symptoms may last only a few days or a few months.

The prognosis is good if the cranial nerves alone are involved, but there is always the possibility of the vital centres being involved, when the prognosis becomes grave.

(2) SPINAL TYPE OF RESPIRATORY FAILURE

This is due to the involvement of the motor cells supplying the respiratory muscles, and may not be clinically obvious for several days after the onset. The diaphragm and intercostal muscles may be affected separately, but one is usually involved to a greater degree than the other.

Grawitz (1896) reported on the first case of an adult with paralysis of the diaphragm due to poliomyelitis. This we found to be present in 11% of our cases.

As long as the temperature is elevated and the acute illness persists, the respiratory muscles must be watched frequently for the earliest signs of impairment of function.

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CEREBROSPINAL FLUID AND BLOOD

CEREBROSPINAL FLUID

IN any large epidemic, there is usually a marked discrepancy in the findings, and naturally the value of lumbar puncture in the diagnosis of poliomyelitis has been much discussed.

The first article emphasizing the diagnostic value of the changes in the cerebrospinal fluid in cases of poliomyelitis was published in 1910 by Flexner and Lewis, and it was followed by a report by Lucas in 1911 on the examination of over five hundred specimens of cerebrospinal fluid.

Dennig and Tartter (1933) advised the examination of the cerebrospinal fluid in doubtful cases, and remarked that in an epidemic period many cases may have fever without any significant changes in the cerebrospinal fluid, and yet are suffering from a very mild or abortive attack of poliomyelitis.

It is more or less an established fact that 10% of all cases show normal findings in the cerebrospinal fluid throughout their illness, but although normal findings may sometimes be found after one lumbar puncture, in the remaining 90% this puncture should be repeated when necessary, as the composition of the cerebrospinal fluid varies according to the different stages of the disease.

Von Kostyal (1933) pointed out that the cerebrospinal fluid reactions in this disease may differ from patient to patient, and as mentioned above, in the same patient from time to time. These findings must therefore be taken into consideration with the other clinical data in an attempt to arrive at an accurate diagnosis.

During the period of the systemic infection, the cerebrospinal fluid is usually normal and remains so during the preparalytic period. Laurinsich (1932) and Gonce (1932) maintain that a diagnosis of abortive and preparalytic poliomyelitis can be confirmed after examination of the

If there is early paralysis of the cervical muscles, the shoulder girdle or upper extremity, there is always the possibility of respiratory paralysis. Again, if there is paralysis of the lower extremities with an accompanying upward extension, then the diaphragm and intercostal muscles are fairly frequently affected.

As respiratory failure sets in, the respirations become increasingly difficult and more rapid and shallow. Dyspnoea is present and the cough reflex is weakened. As the result of the paralysis, the excursion of the chest wall and/or diaphragm is diminished. The patient becomes fatigued, restless, irritable and unable to sleep. Asymmetric and other abnormal but perfectly rhythmical movements of the chest may be noticed.

Later, the respirations become extremely rapid and the accessory muscles of respiration are brought into action. Occasionally the sternomastoid and trapezius are markedly contracted, thus holding the thorax in the position of inspiration and therefore making breathing most difficult. The alae nasae dilate and cyanosis develops, gradually becoming more marked. The patient is disinclined to talk, and there may be twitching at the corners of the mouth, but consciousness is usually retained until the end.

BIBLIOGRAPHY

- GRAWTZ (1896). *Berl. klin. Wschr.*, 33, 245
GUYTON, A. G. (1948). *J Tenn med. Ass*, 41, 254.

of cells during the preparalytic stage. The average count is 50-200 per cmm and it is found that the polymorphonuclear leucocytes predominate in the first few days, to be succeeded later by the lymphocytes which usually outnumber the polymorphonuclears by the end of the second week. In some cases, however, the cell count may be normal at the end of this time, and at this stage the protein content, glucose and chloride levels are almost invariably normal.

In approximately 90% of the paralytic cases, there is an increase in the cellular contents to between 30 and 900 cells per cubic millimetre, but occasionally this may rise to 2,000 cells per cubic millimetre.

During the first week of paralysis the cell count may remain high, but usually by the seventh to tenth day it is starting to fall, and reaches normal about the twenty-first day. Coinciding with this fall in the total number of cells, the percentage of polymorphonuclear leucocytes falls rapidly and that of the lymphocytes rises.

Large mononuclear cells and plasma cells are also occasionally found.

If there are signs of bulbar involvement, the total number of cells is usually small.

Some cases with high cell counts were of the abortive type whilst some with extensive paralysis showed the presence of only a few cells. Some observers argue that a high cell count shows a protective mechanism and that a low count indicates that the resistance of the patient is low, but this is by no means a satisfactory explanation. With the establishment of paralysis, the cells tend to disappear rapidly.

The protein content of the fluid is usually normal in

300 mgm. per 100 cc. of fluid or as high as 800 mgm. It then falls gradually during the next two to three weeks, but it may remain high for as long as six to eight months.

In an excellent paper, Aldelman *et al* (1946) showed that the cerebrospinal fluid protein level was 55-1 mgm per cent in 79% of contact children convalescing from suspected

cerebrospinal fluid, but other observers have been able to do this only in a very small percentage of cases, where the pressure has been increased along with that of the cell count and protein content.

Drury and Sladden (1939) went so far as to state that the finding of a completely normal fluid practically excluded a diagnosis of poliomyelitis, but Pray (1947) found a normal cell count in 50% of his abortive cases and in 15% of the paralytic ones.

During the general infectious period, and the latent period, lumbar puncture is usually negative, but with the penetration of the choroid by the virus and the onset of the meningitic signs, lumbar puncture is usually positive. This is also true if there are signs of an ataxic tremor, slight unrecognized weakness or definite paralysis.

The cerebrospinal fluid must be examined as soon as possible. It is generally agreed that when the diagnosis is unassailable, examination is not required and may in fact be preferably omitted. There is, however, no evidence to show that lumbar puncture in an abortive or non-paralytic case leads to the development of paralysis. Seigl (1934) found that the pressure was increased at the onset of the disease, but this is certainly not a common finding. If the pressure happens to be raised, it usually lies between 150 mm and 200 mm. of water. In 90% of cases the fluid is clear and colourless whilst in the remaining 10% it is faintly opalescent. A fine pellicle formation may appear on standing. This network of fibrin and contained blood-cells may be present within a few days of the onset of the paralysis or if the protein content is over 90 mgm. per cent. It is still unsettled as to whether the pellicle formation depends on the presence of the fibrinogen.

It should be stressed that the cell content of the cerebrospinal fluid should be examined immediately after its collection because the number of polymorphonuclear leucocytes falls rapidly if the fluid is kept.

Most of the recent investigations, including those of Dornedden (1933), Eyre-Brook (1942) and Laurent (1947) find that there is a definite progressive increase in the number

are both increased, and greatly varying degrees of turbidity are found according to the stage of the disease and the treatment which has been given. The protein is increased and also the globulin. Polymorphonuclear leucocytes are usually present in large numbers and the causative organism is very frequently found.

(4) *Tuberculous Meningitis.* The fluid is clear and under an increased pressure. A threadlike coagulum may occur on standing and in it a few scattered tubercle bacilli may be found. The protein is increased but the chloride and sugar content is usually reduced. The cell content is increased from 10 to 400 per cmm. of which usually 75% are lymphocytes.

BLOOD

Most references in the literature state that the virus of poliomyelitis has never been discovered in the blood, but Ward *et al.* (1946) succeeded in isolating it from the blood on one occasion.

It has also been stated that there is no correlation between the clinical types of poliomyelitis, the severity of the paralysis and the elevation of the blood sedimentation rate. Neither does the level of the white cell count afford any guidance

per cmm. and a relatively high polymorphonuclear cell count. Of the white cells, 80% to 85% may be neutrophils. The blood-count is, however, very variable, and in exceptional cases a leucopenia may be present. The monocytes and lymphocytes show no characteristic features and the picture is that usually seen in any other acute infection. Leucocytosis may be present and is usually a lymphocytosis. In one of our cases with a white blood-count of 6,000, 48% were polymorphonuclear leucocytes, 46% lymphocytes and 4% eosinophils. In another case where the white blood-count was 16,000, the eosinophils were 22% and eosinophilia was present for several weeks. Extensive search failed to reveal any explanation.

subclinical poliomyelitis, and that it returned to normal in five to nine weeks after the onset of the illness. Of the paralytic cases, 20% to 25% had normal spinal fluid protein levels in eleven to forty-five days after the onset.

It must be emphasized that any alteration in the number and character of the cells in the cerebrospinal fluid bears no relationship to the type and severity of the disease, nor is there any correlation between the increase in the protein content and the extent of the paralysis.

Always bearing in mind the dangers of repeated lumbar puncture, two or three specimens should be taken during the first three weeks of the illness. A single specimen taken at the end of the second week may give practically normal results, the cell count having returned to normal whilst the rise in the protein content is at this time just commencing.

The majority of workers report an increase of globulin in the cerebrospinal fluid and it frequently remains high for about a month. The chloride and glucose content of the cerebrospinal fluid usually remains normal. The colloidal gold curve is invariably abnormal and shows a rise in the paretic but more so in the leucic zones.

Drury and Sladden (1939) consider that this test has a definite diagnostic value, but a normal fluid finding should not alter the diagnosis if the clinical findings are definite.

Examination of the cerebrospinal fluid may also be helpful in the differential diagnosis of the following four fairly common conditions:

(1) *Landry's Paralysis* The cerebrospinal fluid is in excess and is invariably clear. In some cases there is an excess of albumen and it may be so high that the fluid clots spontaneously.

(2) *Encephalitis Lethargica*. The quantity and pressure of the fluid are increased, the cells are invariably lymphocytes and the protein may be normal or very slightly increased. A fibrin clot is never found in this condition.

(3) *Septic Meningitis*. The pressure and amount of fluid

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Thelander *et al.* (1931) found that the blood-count during the acute phase showed a slight leucocytosis of 10,000–15,000 per cmm. and a relatively high polymorphonuclear cell count. Of the white cells, 80% to 85% may be neutrophils. The blood-count is, however, very variable, and in exceptional cases a leucopenia may be present. The monocytes and lymphocytes show no characteristic features and the picture is that usually seen in any other acute infection. Leucocytosis may be present and is usually a lymphocytosis. In one of our cases with a white blood-count of 6,000, 48% were polymorphonuclear leucocytes, 46% lymphocytes and 4% eosinophils. In another case where the white blood-count was 16,000, the eosinophils were 22% and eosinophilia was present for several weeks. Extensive search failed to reveal any explanation.

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- GONCE, J E (1932) *J Lancet*, 52, 548
KOSTYÁL, L VON (1933) *Arch Kinderheilk*, 100, 15
LAURENT, L J M (1947) *Proc roy Soc Med*, 40, 927
LAURINSICH, H (1932) *Pediatrics (Napoli)*, 40, 38
LUCAS, W. P (1911) *Amer J Dis Child*, 1, 230
PRAY, L G (1947) *J Lancet*, 67, 202
SEIGL, J (1934) *Wien klin Wschr*, 47, 888
THELANDER, H E, *et al* (1931) *Amer. J Dis Child*, 42, 1117
WARD, R, *et al* (1946) *J clin Invest*, 25, 284

Anaemia does not appear to be a predisposing factor to the onset of poliomyelitis, nor does it invariably occur in the convalescent stage.

In poliioencephalitis, a slight leucocytosis and a moderate hypochromatic anaemia have been reported.

The blood sedimentation rate may be normal in uncomplicated cases or it may be raised in 50% of cases at the start of the disease. This elevation makes the test of little help in differential diagnosis, but if the rate is markedly raised the diagnosis is more likely to be that of cerebrospinal meningitis.

Certain observers have attempted to ascertain whether a patient with any particular blood group is more liable to contract poliomyelitis, and it has been stated that patients with blood group O are relatively susceptible, whilst group B patients are normally resistant. My own results compared with other observers are shown in the following table:

TABLE VI

Blood Group	% population in each group	% of cases in each group	
		Taylor	Other observers
AB	3	nil	nil
A	42	55	30
B	9	5	5
O	46	40	65

It would therefore appear that the blood changes in poliomyelitis, although varying from case to case, are of very little diagnostic significance, nor does any particular group appear to be unduly susceptible.

BIBLIOGRAPHY

- ALDELMAN, M. B., *et al* (1946) *Sth med J (Bgham, Ala)*, 39, 706
 DENNIG, H., and TARTIER (1933) *Med Welt*, 7, 1059.
 DORNEDDEN, H (1933) *Gesundh Fürz, Kindesalter*, 8, 216.
 DRURY, J. C., and SLADDEN, A. F (1939) *Brit med J*, 2, 557
 EYRE-BROOK, A. L (1942) *Brit med J*, 1, 758
 FLEXNER, S., and LEWIS, P. A (1910). *J. Amer med Ass*, 54, 1140

fibres as early as the seventh day. The fatty degeneration of fibres was less striking in the later cases in which there was an increase of fibrous tissue with deposits of fat in the connective tissue. There was no evidence to suggest that there had been either fibrotic differentiation or regeneration of muscle fibres in later cases. Early shortening and loss of normal elasticity and resilience are found in the tendinous, ligamentous and fascial structures, e.g. early contractures of the plantar fascia in the tendon sheaths, ilio-tibial fascial tracts, hamstrings, fascial layers in the lumbosacral region and in the periarticular structures of the spine.

Muscle tenderness may be entirely absent in muscles which subsequently become paralysed or there may be the same distribution of tenderness and subsequent paralysis. On the other hand, there may be an equal distribution of tenderness in all the affected muscles, and yet there is an unequal degree of paralysis. It has also been stated that the paralysed muscles may be the most painful. This is especially true in the case of the deltoid which may be later found to show no evidence of recovery.

During the acute stage of the illness which may last from one to six weeks, one of three types of pain and tenderness may be elicited from the muscles:

- (1) Pain due to muscle spasm which is variable in amount.
- (2) Pain elicited on palpation or deep pressure.
- (3) Pain elicited on stretching the muscles.

Each of these three types of pain is quite distinct and care must be taken to differentiate between them.

The muscle pain and tenderness may be localized or have a segmental distribution and it is invariably most obvious in the neck, back and extremities. Pain in the limbs may occur in 10% to 20% of cases—18% of our cases—and may last for days or weeks after the temperature becomes normal. The pain is usually evoked or exaggerated by touch, pressure or movement, and the site of the pain may denote subsequent severe paralysis. Muscles which are the least affected by paralysis may be the most tender, but on the other hand some cases with severe and widespread paralysis are painless.

CHAPTER VII

MUSCLE

MUSCLE involvement is usually more widespread than was at first suspected. It may be accepted that if one muscle is considerably affected, other muscles are nearly always weakened.

The less common muscular signs and symptoms are probably due to virus lesions in the motor cortex, vestibular nuclei, reticular formations or other parts of the extra-pyramidal tracts. These are twitchings, tremors and tenderness which may vary greatly in intensity, and have been observed in some epidemics in 95% of cases.

Muscle Tremor

In 1913 Collivier referred to a peculiar twitching, tremulous or convulsive movement of certain groups of muscles, which lasted for a few seconds to a minute. These muscular tremors are usually present in the upper limb and are seldom seen in adults. If the patient is asked to extend the arm, and to hold it in this position, fine muscular tremors may be noted. The tremor frequently indicates the localization of the paralysis and may precede it by a few hours. Occasionally, however, the tremor may be present but no paralysis develops. In cases with tremor, the voluntary movements are apt to be jerky in nature, and in fulminating cases the tremor may involve the whole body. Jerky movement of the limbs was noted in 22% of our cases, and twitching of individual muscles in 12%.

Muscle Tightness, Tenderness and Pain

According to certain workers there is early evidence of longitudinal splitting of the muscle fibres with a patchy fading of cross striation, alterations of the staining reaction and fatty degeneration within the fibres. Cellular infiltration was conspicuous and necrosis was seen in isolated muscle

severe pain with slight paralysis in 3 cases. In the remaining 2 cases both pain and paralysis was slight.

Muscle Spasm

The apparent confusion on this subject in the literature appears to be due to the different conceptions of various observers as to what actually constitutes muscle spasm.

In our opinion, there are two types of muscle spasm. The first type is the early spasm of the acute stage which lasts for a few days. This transitory spasm is never a therapeutic problem as it usually disappears spontaneously. It is relieved by moist heat, or by sympathetic paralysants. One observer noted that the frequency and distribution of muscle spasm as noted clinically in his cases was as follows:

TABLE VIII

Muscle in spasm	% of total cases
Posterior muscles of neck	100
Posterior spinal muscles	100
Hamstrings	100
Calf muscles	80
Tensor fasciae femoris	53
Upper trapezius	20 8
Plantar fasciae	14 5
Quadratus fem	9 7
Pectoralis major	9 3
Quadratus lumb.	6 6

The second type is the spasm which persists for more than a month, and it is most troublesome as it may interfere with the general treatment of the patient. It is a reflex contracture due to a pain stimulus, and it is frequently present along with stiff joints and contractures. It is only present if there is a limitation of movement in a particular limb.

The various theories of the causation of muscle spasm may be summarized thus:

- (1) Irritation of the anterior horn cells in the spinal cord.
- (2) Structural and functional changes in the internuncial

throughout. The pain may be persistent, and may indicate an abnormal condition within the affected muscles, but apparently healthy muscles may also be acutely painful and tender. Contracted muscles when put on the stretch may be painful, and many of them have no voluntary contractile power. The experience of observers in this country has shown that pain and tenderness on stretching a muscle group was not a common finding, and was usually observed only after the paralysis had been well established.

The pain has been attributed to—

- (1) Meningeal irritation.
- (2) Involvement of the spinal ganglia of the posterior roots.
- (3) Ischaemia due to vasoconstriction from involvement of the central nervous system, or a muscular contraction of the walls of the blood-vessels.
- (4) Lesions of the brain stem.

Muscle tenderness lasted in the majority of our cases for periods varying from one day to one week and in the minority it lasted up to five weeks. This is shown as follows:

TABLE VII

Time in days	No. of cases	Time in weeks	No. of cases
1	10	1	81
2	23	2	19
3	14	3	11
4	10	4	1
5	9	5	1
6	1		
7	14		
Total	81		

Degree of Muscle Tenderness

This was particularly noted in 16 cases. There was slight tenderness with severe subsequent paralysis in 5 cases. The muscles of the legs and arms were very tender, but there was no resultant paralysis in 6 cases, whilst there was moderately

ship between the degree of paresis and the frequency and intensity of the spasm. These observers are also of the opinion that spasms may be due to vasomotor disturbances

in the antispasmodic drugs even if they do relieve muscle pain and spasm. On the other hand, there is no definite proof that the relief of muscle spasm prevents paralysis or alters the mortality in the acute stage.

One of our cases of muscle spasm was treated by a parasympathetic block with an immediate increase in the temperature of the left leg which became more relaxed, and passive movements became possible. Spasticity returned on the second day, but after manipulation it relaxed, and by the end of the first week a full range of passive movements was obtained.

Observers who have seen a great deal of muscle spasm state that in the early stages it may be present in the weakened muscles, their antagonists, and also in muscles which do not show the slightest evidence of paresis. Another important recent observation is that in poliomyelitis the contraction of an affected muscle is not accompanied by a relaxation of its antagonist. In one of our cases, spasm of the psoas muscle was followed a week later by a flaccid paralysis. This case was first diagnosed as a tuberculous hip joint.

The incidence of muscle spasm appears to differ greatly in different countries, and in this country has so far been rare except as a manifestation of meningeal irritation.

According to Pollock *et al.* (1947), muscle spasm is usually absent during the time of the development of the paralysis, but when present it varies greatly in its degree and duration. In the preparalytic stage, spasm temporarily impairs muscle function and destroys the normal reciprocal relation between agonists and antagonists. Pohl (1947), however, stated that spasm appears early and is present in every case. It is generally more severe in the paralytic patients, and the longer it is untreated, the more difficult it is to relieve. When com-

neurones and reflex irritability of the anterior horn cells

- (3) Involvement of the posterior spinal ganglia.
- (4) Lesions in the brain stem
- (5) Stretch reflex.
- (6) Protective pain reflex.
- (7) Peripheral pain causing hyperexcitability in the proprioceptive reflexes.
- (8) Ischaemia which results in pain and reflex rigidity of the skeletal muscles.
- (9) Direct action of the virus on the muscle fibres or on the myoneural junction.
- (10) Disturbance of the acetylcholine esterase mechanism of the peripheral neuromuscular junction.

Certain observers have produced histological evidence which suggests that involvement of some of the upper motor neurones may be responsible for the hyperirritability of the spinal reflex centres. Structural abnormalities in the cells have been found in the motor cortex, vestibular nuclei and cerebellar nuclei. Involvement of the pyramidal and extra-pyramidal systems may also account for some of the reflex hypertonicity.

Schwartz and Bouman (1942) found widespread hyperirritability on stretching the muscles in spasm and their apparently healthy unaffected opponents. Moldaver (1943) reported that spasm was due to a result of the combination of the normal stretch reflex, meningeal irritation of the posterior roots, increase in the normal tone of the normal muscles opposed to the weak or paralysed muscles, and to lesions of dorsal root ganglia and posterior horns.

Bodian (1946) is of the opinion that muscle spasticity is a reflex phenomenon associated with increased stretch reflexes. Widespread spasticity may be found to affect the extensor and flexor muscles whether they are of normal strength or weakened.

As Luft and Muller (1947) point out, the occurrence of spasm does not appear to depend on the degree of injury to the peripheral motor neurones, and there is no relation-

secondary to lesions in the inhibitory area of the brain stem. The vestibular formations are affected so that the control of the antigravity muscles is disturbed resulting in hyperactivity or spasm.

Kernig's sign is positive in a large number of cases, but it is frequently absent in infants. In 20% of our cases, this sign was positive, and head retraction was present in 18%.



FIG. 5 — AMOSS'S SIGN

(Reproduced by permission of the Nursing Mirror)

A stiff back due to muscle spasm, which occurred in 62% of our cases, may be diagnostic if other local causes can be excluded. If the patient is placed in a sitting position, he will support himself by placing his hands behind him in the so-called tripod position (Amoss's sign). Difficulty in maintaining the sitting position may be due to spasm or shortening of the hamstring muscles, and if the patient is made to sit with his knees flexed over the edge of the bed, it will be found that he can straighten his back fully. If, whilst in this

bined with paralysis, it contributes to the fixed position and produces secondary contractures.

As Richards *et al.* (1947) pointed out, some patients during the first few days complain of a very slight stiffness of the neck and back muscles which later increases and may or may not be accompanied by paralysis. It may be of minimal intensity and disappear rapidly when the inflammatory process in the cord subsides. It may, on the other hand, spread



FIG. 4—KERNIG'S SIGN

extensively to other parts of the body and persist for weeks or months whether treatment is given or not. The presence of peripheral encephalitis, or irritation of the dorsal nerve root ganglia and meninges, and all degrees are met with. It was present in 22% of our cases. The head can be flexed without any resistance through a fairly large arc, whereas in meningitis the head is definitely retracted.

Recently it has been suggested that muscle spasm is

the effect of gravity disappears, the anterior neck muscles bring the head forward into a neutral position.

Spasm of the respiratory muscles may lead to marked dyspnoea, whilst spasm of the abdominal muscles may simulate appendicitis.

Careful investigations of muscle spasm seem to show that it does not initiate the development of muscle weakness. Spasm, however, has been said to impair the muscle function, and its relaxation reduces the number of, or completely prevents deformities. This relaxation can therefore in itself be considered a sign of improvement.

Muscle spasm may cause contractures and deformities by muscle imbalance, and if not adequately treated there is loss of function, contraction, fibrosis and muscular atrophy. Pain and limited movement even after several weeks of treatment may be due to fibrosis, contractures or adhesions. These are the main causes of muscle shortening and should be treated by passive stretching by an experienced physiotherapist. Muscle spasm is aggravated by massage, premature weight-bearing and exposure to cold.

Paresis and Paralysis

In our series, 50% of cases were of sudden onset, and in the other 50% the onset was gradual.

Muscular weakness in poliomyelitis has been attributed to one or other of the following causes.

- (1) Pathological changes in the nerve cells and fibres. Temporary paralysis may be due to inflammatory and toxic changes, whilst permanent paralysis is due to the destruction of the nerve cells.
- (2) Mechanical strain
- (3) Overfatigue resulting from overactivity or over-treatment.
- (4) Muscle stretch weakness.
- (5) Disuse atrophy

It has been stated that the apparent weakness or paralysis seen during the acute stages of poliomyelitis is the sum of at least three different effects.

position, he is asked to touch his knees with his nose or lips, he is unable to do so. In a few cases the back rigidity produces such severe pain that even slight flexion is impossible. The patient therefore assumes the position of opisthotonos, and if the spasm of the neck and back muscles is prolonged, it may result in considerable shortening of the muscles in this position.

Spasm of the posterior muscles may also be demonstrated



FIG 6—KNEE-KISSING TEST

by flexing the head and neck of the patient whilst he is lying in the supine position. This will cause flexion of the legs (*Brudzinski's sign*)

Another important sign is that of *Ruhrah*. This is elicited by lifting the patient's shoulders off the bed. Normally the head should remain in the same plane as the trunk, but in poliomyelitis the head falls back and remains in this position until the trunk is nearly erect. The explanation given is that there is imbalance of the neck muscles caused by hypertonicity or spasticity of the posterior group, which aided by gravity cannot be overcome by the anterior group. When

without any detectable weakness. There is, however, a close correlation between the severity of the paralysis and the number of motor neurones destroyed. Evidence of this may be demonstrated histologically in a patient who has died from respiratory or cardiac failure.

The region of the spinal cord from which the nerve to the affected muscles arises may be very small, or it may extend over a relatively large area. The severity of the paralysis would therefore appear to depend on the extent to which this area is involved, because if all the individual nerve fibres within the nerve trunk are not affected, the muscle will be only partially paralysed. This is in contrast to what one would expect if the whole nerve trunk were involved in every case.

The paralysis may also be masked by the presence of muscle pain and tenderness, or the paralysis may be considered to be much more severe than it really is, owing to a reluctance on the part of the patient to move the limb because of pain.

Involvement of the spinal cord results in a weakness or flaccid paralysis of one or more of the muscles of the thorax, abdomen, back, and upper and lower extremities. This paralysis may affect a few of the muscle fibres in a muscle, any one particular muscle, a group of muscles or any combination of muscle groups in either the arm or the leg. In the more severe cases, it may affect the whole arm or leg, or any combination of the four limbs, with or without involvement of the muscles of the trunk. We therefore see that the muscular signs vary from those of minimal weakness to those of complete paralysis. It is always essential to examine and compare the muscles of each side, because in

doubtedly the most common finding. Seddon later stated that the lumbar enlargement was involved in 86.3% of his

found that the left leg was more often affected than the right.

- (1) Pseudo-paralysis—resulting from pain and tenderness
- (2) Temporary paralysis resulting from temporary loss of function of the anterior horn cells which can recover without axonal degeneration. Tested electrically, the muscles do not show the reaction of degeneration.
- (3) Permanent paralysis resulting from irreversible damage to the anterior horn cells.

It was also pointed out that a patchy distribution of muscle weakness is due to a combination of muscle weakness and muscle spasm.

The extent of the paralysis appears to depend upon:

- (1) The resistance of the patient.
- (2) The virulence of the particular strain of virus present.
- (3) The dosage of the virus.
- (4) A history of recent trauma, e.g. tonsillectomy or adenoidectomy.
- (5) Chilling and recent excessive exercise.
- (6) Pregnancy

It should be noted that the paralysis seldom spreads for more than about three days. During this period of progressive paralysis, the patient must be carefully observed for any evidence of respiratory paralysis due to bulbar or spinal involvement. If this complication arises, there is continued fever, and a slow and progressive paralysis up to the time of death.

The muscles appear to be involved in proportion to their activity or fatigue, and experience has shown that the most highly developed muscles are those which are the most severely affected, e.g. the forearm muscles in a violinist.

A muscle cannot regain full effective power if its synergists and fixators are paralysed. The maximum tests cannot be applied to muscles like the calf and anterior tibial, unless the patient is asked to walk on the toes or heels.

Paresis may be present but not observed because the destroyed motor neurones are widely scattered and thus a single functional motor group is not involved. It has been shown that one-third of the motor neurones may be destroyed

the abdominal and back muscles, the thoracic muscles, and finally the neck and respiratory muscles. The paralysis may, however, halt at any stage, and then show signs of regression, and it may eventually leave the patient with only a very slight residual paralysis.

In our series the ascending type occurred in 30% in the 5-15 age group and 45% in the 0-5 group. The descending type was found in approximately 60% in all age groups.

In the ascending and descending types, the progress of the active disease may proceed for several days, but it is occasionally interrupted by a period of remission. In our series, 80% occurred in the first twenty-four hours. A most peculiar



FIG. 7.—BREVOR'S SIGN

type of remission is seen where the limbs are involved one at a time at intervals of approximately twelve hours, the maximum spread having occurred in about four days. This type of case may end fatally from the involvement of the respiratory muscles.

The abdominal muscles may be affected in 80% of paralytic cases, and this is usually bilateral. In cases of paralysis of the lower abdominal muscles, if the head is raised, the umbilicus moves upwards. This indicates a lesion at the level of the tenth intercostal nerve (Brevor's sign).

If the spinal muscles are involved and especially if this is combined with an asymmetrical affection of the abdominal muscles, a special watch must be kept for spinal curvature.

Usually the paralysis gradually diminishes, the distal muscles recovering more quickly and more completely than

There is no doubt that the legs are affected about twice as often as the arms, and that the paralysis is usually more severe. The right arm was affected five times as frequently as the left. Hemiplegia was not a common finding, but it was twice as frequent on the right side. All four limbs and trunk were affected in three cases. The anterior tibial, peronei, quadriceps and gluteal muscles tend to be constantly and severely affected. The proximal muscle groups are said to be more affected than the distal, and this was true in our cases. The extensors of the knee, hip and foot are more often affected than the flexors. The abductors of the hip are more often affected than the adductors and the evertors of the foot more often than the invertors. The hip abductors seem to be more prone to bilateral involvement than the tibialis anticus, soleus and peroneal muscles.

If the muscles of the foot and toes are paralysed, the plantar responses are flexor or absent. Alterations in, or loss of the reflexes does not necessarily mean that paresis or paralysis is present, but this is much more probable if the reflexes are asymmetric. A pseudo-Babinski sign may result from paralysis of the flexor muscles whilst the extensors retain their power of contraction.

Cases may occur with paralysis of one or both upper limbs, and in which the knee or ankle reflexes or both may be diminished or absent, and yet there is no sign of muscle weakness in the lower limbs. If these cases are followed up for a considerable time, it will be found that the reflexes recover fully in a large percentage of cases.

The arms are more frequently involved than the trunk, and the proximal arm muscles are affected more often and more severely than the distal ones. The right arm appears to be affected twice as often as the left. The deltoid is the muscle most frequently involved, the next in frequency being

or diaphragmatic paralysis.

If the spread of the paralysis is of the ascending type, it usually commences in the legs and involves progressively

be perfected by a prolonged and painstaking personal and practical experience, but it is earnestly hoped that the following observations will prove to be helpful.

If the patient is acutely ill, accurate muscle testing is impossible and unjustifiable. An experienced observer, however, should be able to obtain a reasonably accurate picture of the degree and extent of the muscle involvement, and this will allow adequate splinting to be applied at the earliest moment.

It has been stated that if an attempt is made to test all the muscles in one session, the patient will become exhausted long before the test is completed, and therefore the later results will be inaccurate. In the early stages of the disease a more accurate assessment will therefore be obtained if the muscle testing is spread over two to three days. In our unit all patients except small children are tested in one session, and we have not observed any detrimental effect attributable to this practice. The picture may of course be obscured by the presence of excessive pain and muscle spasm.

On admission, each case should have a chart of the whole muscular system completed, otherwise a weakened muscle may inadvertently be missed. This chart, which is part of the diagnostic procedure, forms the keystone of the rehabilitation programme, and the whole course of treatment to the muscles is based upon its accuracy. It should be stressed that frequent thorough muscle testing might increase the paralysis, and should be avoided.

The muscle chart shows the extent of the original involvement and the rate and degree of the return of the muscle power. It therefore helps to determine the prognosis, always bearing in mind the interval between the onset of the disease and the initiation of adequate treatment.

The system of grading used by us is that recommended by the Peripheral Nerve Injuries Committee of the Medical Research Council, and this gives the following valuations:

- 0 = no contraction present.
- 1 = flicker of movement which can be seen and felt.
- 2 = muscle contraction with gravity eliminated
- 3 = muscle contraction against gravity only

the proximal ones. Clinically some of the muscles are permanently paralysed and finally degenerate in whole or in part, depending upon the degree of damage to their controlling motor nerve cells. These completely paralysed muscles show the reaction of degeneration, but the degeneration of a muscle is a very slow process. Other muscles undergo disuse atrophy, whilst their controlling motor nerve cells are temporarily out of action; but with the resumption of the normal function of the cells, even very weak muscles may recover reasonably good or normal strength.

The return of muscle strength which occurs from one day to two years has been attributed by various observers to:

- (1) Destruction of the virus and recovery of function of the nerve cells.
- (2) Hypertrophy of the remaining normal muscle fibres.
- (3) Muscular development of the involved muscles by appropriate therapy
- (4) Muscular development after correction of deformity and relief of strain.
- (5) Re-routing of pathways in the brain stem.
- (6) Trick movements.

It will therefore be appreciated that the muscle picture in *poliomyelitis* is very complicated in the early stages when we have paretic or paralysed muscles, or muscles in spasm. Impulses from these muscles may pass to other muscles supplied by nerves arising from the same level of the spinal cord.

MUSCLE TESTING AND CHARTING

It is assumed that the reader already knows the origin, insertion, nerve supply, action of the muscles, as well as their contraction and overaction.

In order to eliminate the human errors in assessing muscle power, certain observers have recommended various mechanical devices for measuring the degree of paralysis, but in this country the majority of centres appear to favour manual muscle testing.

The testing of voluntary muscle power in muscles can only

ELECTROMYOGRAPHY

In the present state of our knowledge one cannot diagnose poliomyelitis or any disease purely by electromyographic observations, but they may be of assistance in establishing the prognosis in poliomyelitis.

The following limitations of this method of investigation must, however, be borne in mind when dealing with poliomyelitis. After insertion of the needle into a muscle belly, the action potentials shown may represent only a very small area, approximately 1 cm. in diameter, of the muscle tissue in close proximity to the needle. We may therefore get a normal response from a paralysed muscle in which there are still a few normal muscle fibres. To assess the excitability of a muscle accurately, it becomes necessary to insert the needle into several different areas of the muscle belly, and this can be a very painful procedure.

Electromyographic exploration shows no electrical activity in normal muscle at rest, but a very complex disturbance during exertion. The complex wave form is due to motor units coming into activity asynchronously, and their action potentials may cancel out or add up. In the early stages of poliomyelitis, although a muscle may appear to be completely paralysed, this particular muscle may show electromyographically that irregular action potentials are being produced when it is apparently at rest, and that it responds rapidly to any movement which causes the patient discomfort.

After two to three weeks fibrillation shows itself as small repetitive spike potentials. This occurring in a muscle at rest is diagnostic of motor unit denervation in a muscle, and results from the autonomous activity of individual fibres from damage to the anterior horn cells. In maximum exertion there is a large reduction in the number of motor units involved. After recovery the muscle or muscle group may show marked wasting, absence of reflex responses and rapid fatigue on exertion.

In electromyography the voltage of the action potentials has been found to give a good index of the contractile

4 = *muscle contraction against gravity and resistance.*

5 = *normal muscle contraction.*

(Examples of useful types of muscle charts are given in the appendices)

The tests are made by giving as much assistance as possible to the muscle being tested, and it is wiser to underrate rather than to overrate the power of a muscle. It need hardly be mentioned that it is always the primary action of the muscle or single group of muscles which is tested. If it is impossible to test an individual muscle, then a single muscle group containing it may be taken and recorded.

In very young children or infants, accurate manual muscle testing is impossible, and only a very rough estimate can be obtained. The patient may lie with the lower limb completely immobile, but if complete paralysis is not present he will usually withdraw the limb when the sole of the foot is tickled. The movements of the upper limb can be tested roughly by holding a toy in different positions in front of the child and asking him to reach for it.

Certain observers have stated that it is essential that the same observer should undertake the muscle testing of a patient from the time of admission and again after an interval of two weeks. The same observer should subsequently repeat it monthly for six months, and then every second month for a year or so, depending upon the particular case in question. In our unit we do not insist on the tests being carried out by the same person, but he or she must be a member of the poliomyelitis team. As a result, we find that the records tally very well, and in addition one observation is always a check on another.

From the muscle chart a preliminary estimate of the extent and distribution of the major muscle weaknesses and an assessment of the factors producing deformity can be arrived at. Hyperaesthesia, muscle tenderness and spasm should also be noted. As passive movements can also be used to determine whether limitation in movement is due to muscle weakness or to some other predisposing factor, the ranges of these movements should always be recorded.

- (2) Muscle spasm is found in parts of the body in which no clinical symptoms are evident.
- (3) Spasticity is found to be reflex in nature and is not present in completely paralysed muscles.
- (4) When the power of voluntary contraction increases during treatment, the spasticity of the muscles decreases

SQUARE WAVE VOLTAGE STIMULATION AND NERVE CONDUCTION

The state of innervation of muscles can determine their threshold responses. The tests are time-consuming and are not used to any great extent in poliomyelitis because the condition here is that no muscle is completely denervated and the interpretation of results has proved unreliable. It has been demonstrated recently that the threshold response of a muscle affected by poliomyelitis is often lower than a normal muscle.

NERVE CONDUCTION

Nerve conduction has been found to have a very useful place in assessing the recovery of muscles and is a valuable guide in the treatment. It is now possible to tell at an early stage whether muscle re-education is worth while.

BIBLIOGRAPHY

BODIAN, D (1946) *Proc Soc exp Biol (N Y)*, 61, 170
 COLLIER, I. A. (1913) *J Amer med Ass*, 66, 912

POLLOCK, L. J., *et al* (1947) *Trans Amer Neurol Ass*, 72, 57

RICHARDS, R., *et al* (1947) *Proc Mayo Clin*, 22, 31

SCHWARTZ, R. P., and BOUMAN, H. D. (1942) *J Amer med Ass*, 199, 93

SEDDON, H. J. (1943) *Lancet*, 2, 549.

TAILLENS, J. (1937) *Rev méd Suisse rom*, 57, 561.

ZOLLIKOFER, R., and CUSTER, H. (1932) *Schweiz med Wschr*, 62, 129

power and to correlate well with the manual and ergographic tests. The electromyogram also gives valuable information as to the degree, distribution and nature of muscle irritability. Electromyographic records show that spontaneous electrical discharges are not present in relaxed muscles and are frequently not demonstrable in spastic muscle. Spontaneous electrical activity has been found more commonly in weakened muscles than in muscles in spasm, and this type of electrical activity is regarded as an index of weakness rather than one of spasm.

In patients with considerable weakness or muscular pain, active muscular contraction showed a disturbance of excitability of the antagonistic muscles, and synchronous diphasic spike potentials coming from both muscle groups were found frequently during the voluntary effort. This abnormality decreased as the muscle strength improved with muscle re-education or after therapy for the relief of pain. The finding of spontaneous electrical discharges from resting muscles during the convalescent period shows that the muscle is recovering. These discharges have the same patterns as those recorded from a muscle whose nerve is known to be regenerating after a peripheral injury. Their appearance coincides with improved electrical excitability and increased muscle power.

If a patient has had careful treatment for six months from the date of infection and there is no sign of the return of voluntary power, it is most unlikely that the muscle will regain any useful function. Careful electromyographical studies of superficial muscle confirm this and show that permanent paralysis can be foretold as early as four to six weeks after the onset. Axonal regeneration does not recur if the reaction of degeneration is obtained. It is probable that careful testing may lead to the early abandonment of useless physiotherapy and advance the commencement of the orthopaedic programme and rehabilitation.

Following electromyographic studies, Schwartz and Bouman (1942) reported their conclusions as follows:

- (1) Spasticity exists in the weakened muscles and their antagonists.

- (2) The majority of the anterior horn cells are seriously damaged but a few may escape. Atrophy is incomplete and during the following two to three years, the muscles will partially recover their function, but they will always remain permanently weakened and atrophic
- (3) The anterior horn cells are completely destroyed and the muscle fibres are replaced by fibrous tissue.

Although limps due to paralysis of individual muscles are frequently described, they are rarely seen. Limps when present are usually of a complicated nature, being due to the paralysis of several muscles.

Having made these few observations, let us now deal with the different regions of the body in the following order:

- (1) Muscles of the spine and trunk.
- (2) Muscles of the lower limb.
- (3) Muscles of the upper limb.

(1) MUSCLES OF THE SPINE AND TRUNK

(1) Flexors of the Spine

Superficial group—sternomastoid and suprahyoid.

Deep group —longus colli, longus capitis, scaleni and rectus capitis anterior

To test the sternomastoids, ask the patient to raise the head and turn first to one side and then to the other. Then ask him to lift the head from the bed and hold the position. Paralysis of the neck flexors can cause a cervical concavity, the extensors become shortened and the patient gets a "poked" neck.

If there is paralysis of the sternomastoids, anterior scaleni, infra and supra hyoids, clavicular portion of the trapezius and the anterior vertical flexors of the neck, the patient is found to be incapable of flexing the cervical spine against resistance. A limited movement may, however, be possible if any of the above muscles have escaped or are only partially paralysed. If the other flexors of the spine are very weak, the patient may be able to flex the cervical spine, but the

CHAPTER VIII

NOTES ON INDIVIDUAL MUSCLE PARESIS OR PARALYSIS

BEFORE discussing this section in detail, let us first of all consider one or two points regarding the muscles themselves.

We know that the movements of individual muscles are not represented in the cerebral cortex and that when a movement is carried out, a particular combination of muscles is brought into activity. It is therefore quite impossible for the individual voluntarily to introduce or subtract a specific muscle from any definite muscular movement.

We also know that a muscle may be the prime mover of only one joint even although theoretically it is capable of acting on every joint over which it passes.

If a prime mover is voluntarily contracted, this will be accompanied by a contraction of the fixation muscles, and a corresponding relaxation of the antagonists. The best example of this is seen when the patient abducts his arm, because this movement is always related to a contraction of the muscles which are inserted into the scapula.

In order that any movement is carried out in the most efficient manner, there must be complete co-ordination of the prime movers, antagonists, fixators and synergists. To illustrate this point let us take the example of the clenched fist, where we know that if the extensors of the wrist are paralysed, the wrist joint will be unstable and the gripping power of the hand consequently weakened.

In poliomyelitis, paresis or paralysis of an individual muscle is very rarely seen, and it is much more common to find that one or more muscle groups are affected.

It is also found that the paralysed muscles fall into one of three categories

- (1) Those in which the anterior horn cells are only slightly and temporarily damaged. The muscles, if they do waste, will do so only temporarily and they will recover their full function within a few weeks or months.

walk with the hips flexed and with the spine in marked lumbar lordosis. The abdomen may be protuberant and the gait waddling in character

If the paralysis of the lateral abdominal muscles is unilateral, and if the weight is borne on the sound side, then the pelvis on the affected side will be seen to drop, thus giving rise to a positive Trendelenburg sign. This sign is most marked if the quadratus lumborum is also involved. In long-standing cases this may be accompanied by a slight adduction contracture and a weakness of the abductors of the opposite side. Paralytic scoliosis may also develop several years after the acute attack and it may progress even after the patient's growth has ceased.

(2) Extensors of the Spine

These muscles are the splenius capitis, semispinatus capitis and erector spinae. If the patient lies in the prone position and the legs are firmly held, it will be found that if the paresis or paralysis of the extensor muscles is marked, he cannot extend the dorsal spine or hyperextend the lumbar or cervical spine. Later it will be found that walking or even sitting erect without a spinal brace is impossible. The flexors of the spine are found to be contracted and there is a dorsolumbar kyphosis. In the less severe cases, the gait is found to be awkward.

In cases with unilateral paralysis of the extensors of the spine, or where the erector spinae are unequally involved, scoliosis usually results with the convexity towards the weaker side. The muscles and ligaments in the concavity are found to be contracted like a bowstring. It has been found that this deformity may develop even when the patient is still recumbent, but the majority of cases occur only after walking is resumed.

(3) The Respiratory Muscles

Paralysis of the diaphragm and intercostal muscles is discussed in the section dealing with paralysis of the respiratory muscles, but the following points should also be noted.

lower portion of his thorax is depressed and the pelvis becomes tilted so that the normal lumbar curve is lost.

The rectus abdominis which is also a flexor of the spine, may be affected alone or along with the other flexors. These muscles are usually affected asymmetrically and any voluntary contraction will show that their power is unequally distributed. In bilateral paresis, the patient may not be able to raise his head and shoulders from the bed without using his arms.

The lateral flexors of the spine may also be paralysed, and to test for this the patient should be placed on his side, when it will be shown that he cannot raise his shoulders from the bed by sliding his hand down the lateral aspect of his thigh.

In paresis of the postero-lateral fibres of the external oblique muscles, the chest will be found to rotate on the pelvis, just before the trunk is fully flexed. If the paralysis is unilateral, the chest wall is found to be rotated posteriorly on that side, whilst if the paralysis is bilateral, the pelvis becomes tilted anteriorly, and there is a marked lordosis.

When there is unilateral paralysis of the internal oblique muscles, this is accompanied by a forward rotation of the thorax on the same side. In bilateral paralysis, if the patient lies in the supine position, it will be found that he is unable to produce flexion of the thorax on the pelvis.

If there is unilateral weakness of both the internal and external oblique muscles, then a scoliosis with the convexity of the curve on the same side will be produced.

Paresis of the transverse abdominal muscles alone produces no effect on the relationship of the pelvis to the thorax, but it could produce a marked protuberance of the anterior abdominal wall. Gross abdominal weakness becomes obvious by a bulging of the abdominal wall on coughing, crying or by any action which increases the intra-abdominal pressure.

Lateral deviation of the umbilicus from the affected side when the patient lifts his head from the bed is said to be a good prognostic sign.

Paralysis of the three lateral abdominal muscles may give rise to later signs when the patient reaches the late convalescent or residual stage. The patient is seen to stand or

walk with the hips flexed and with the spine in marked lumbar lordosis. The abdomen may be protuberant and the gait waddling in character.

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(3) The Respiratory Muscles

Paralysis of the diaphragm and intercostal muscles is discussed in the section dealing with paralysis of the respiratory muscles, but the following points should also be noted.

It should be mentioned that respiration in the first two years of life is almost entirely diaphragmatic. In diaphragmatic paralysis where the intercostal muscles escape, the respirations are entirely thoracic and paradoxical in nature, i.e. on each inspiration the diaphragm rises and there is an epigastric retraction, due to the suction on the paralysed diaphragm from the negative intrapleural pressure and on expiration the opposite effect takes place. If firm pressure is applied to the sides of the chest, the patient becomes markedly distressed and sometimes cyanotic. Mackenzie states that the abdominal muscles are antagonists of the diaphragm. When it is necessary to palpate the abdomen, reciprocal relaxation of the abdominal muscles can be obtained by asking the patient to draw a deep breath.

In bilateral paralysis of the intercostal muscles, the breathing becomes mainly diaphragmatic and there is an alternate bulging and retraction of the intercostal spaces. Pressure on the abdomen causes marked respiratory distress.

Weakness of the respiratory muscles is often accompanied by coarse râles in the chest and this paresis frequently predisposes to broncho-pneumonia.

(2) MUSCLES OF THE LOWER LIMB

As mentioned previously, it is more usual for a particular group of muscles to be attacked than an individual muscle. I think that a brief revision of the various muscles which produce the movements of the joints together with a description of the results of paresis or paralysis of the principal muscles would be most useful. This should enable the reader to determine the various muscles or muscle groups involved and to analyse any abnormality in the gait.

(A) Movements of the Hip Joint

(1) *Flexion.* The range is from 0° to 120° , and the principal muscle which carries out this movement is the ilio-psoas. The accessory muscles are the rectus femoris, pectineus, sartorius, adductor longus, adductor brevis, and the pubic part of the adductor magnus. It is limited by contact of the

thigh with the anterior abdominal wall, but if the knee is extended, it is limited before this occurs by the tension of the hamstring muscles. It has been suggested that the sartorius, adductor longus, and pectineus probably flex the hip to the horizontal position, when at that point psoas takes over the action. As the action of the ilio-psoas usually varies according to the position of the limb, a paralysis of this muscle will result in a weakness of these movements. We therefore see that such a paralysis will produce difficulty in flexing the extended thigh and also a weakness in medial rotation. The thigh tends to rotate laterally during flexion of the hip joint, and if the limb is placed in full flexion, there is a weakness in lateral rotation. Paralysis of the psoas may cause a strong tensor fasciae latae to hypertrophy. If there is paralysis of the tensor fasciae latae, flexion may be carried out in lateral rotation, because tensor fasciae latae is a medial rotator as well as a rotator.

Unilateral paralysis of the psoas may cause scoliosis.

If the lateral abdominal muscles are strong, they may substitute in flexion of the hip, although inadequately.

Tests for hip flexion must exclude abduction and medial rotation.

In bilateral paralysis of the flexor muscles of the hip joint in the residual stage of the disease, there is an anterior displacement of the pelvis and a lumbar kyphosis. The patient will complain of difficulty in assuming the sitting-up position from recumbency, or the erect position after sitting on a chair.

In walking, the leg on the paralysed side can be brought forward only by rotating the whole pelvis on that side and dragging the leg forwards. It will also be noticed that the opposite leg appears to be medially rotated, and in unilateral cases lumbar scoliosis may be present. Walking or climbing stairs is found to be extremely difficult.

If the lateral abdominal muscles escape, they are found to substitute for the paralysed flexor muscles of the hip joint, but they do so very inadequately.

(2) *Extension.* The range of movement is from 0° to 15° , and the principal muscle performing this movement is the

It should be mentioned that respiration in the first two years of life is almost entirely diaphragmatic. In diaphragmatic paralysis where the intercostal muscles escape, the respirations are entirely thoracic and paradoxical in nature, i.e. on each inspiration the diaphragm rises and there is an epigastric retraction, due to the suction on the paralysed diaphragm from the negative intrapleural pressure and on expiration the opposite effect takes place. If firm pressure is applied to the sides of the chest, the patient becomes markedly distressed and sometimes cyanotic. Mackenzie states that the abdominal muscles are antagonists of the diaphragm. When it is necessary to palpate the abdomen, reciprocal relaxation of the abdominal muscles can be obtained by asking the patient to draw a deep breath.

In bilateral paralysis of the intercostal muscles, the breathing becomes mainly diaphragmatic and there is an alternate bulging and retraction of the intercostal spaces. Pressure on the abdomen causes marked respiratory distress.

Weakness of the respiratory muscles is often accompanied by coarse râles in the chest and this paresis frequently predisposes to broncho-pneumonia.

(2) MUSCLES OF THE LOWER LIMB

As mentioned previously, it is more usual for a particular group of muscles to be attacked than an individual muscle. I think that a brief revision of the various muscles which produce the movements of the joints together with a description of the results of paresis or paralysis of the principal muscles would be most useful. This should enable the reader to determine the various muscles or muscle groups involved and to analyse any abnormality in the gait.

(A) Movements of the Hip Joint

(1) *Flexion.* The range is from 0° to 120° , and the principal muscle which carries out this movement is the ilio-psoas. The accessory muscles are the rectus femoris, pectineus, sartorius, adductor longus, adductor brevis, and the pubic part of the adductor magnus. It is limited by contact of the

and if the anterior fibres are mainly affected, medial rotation. If the posterior fibres are affected, lateral rotation is weaker.

Paralysis of the gluteus minimus is found to produce similar lesions to those of a paralysis of the gluteus medius.

Later, when the patient puts his weight on the affected side, the pelvis suddenly lurches to that side, and in an attempt to regain his balance, the patient immediately extends his arm laterally. The reason for this is that in walking the function of gluteus medius is to raise the pelvis by the leg stepping forward to clear the ground. With unilateral paralysis the pelvis droops on the side of the weakness, and the body sways to that side to maintain balance. This produces a characteristic limp which cannot be corrected by any appliance or by building up the sole of the boot. If the tensor fasciae latae is strong on that side, it may be trained as a hip abductor in extension. Experimentally the limp can be corrected by a weight held in the hand on the affected side, as this restores the stability by replacing the absent downward muscle pull.

In bilateral paralysis, the patient must swing his body from side to side in order to place one foot in front of the other.

(4) *Adduction.* The range of movement is from 45° to 0° , and the principal muscles producing this movement are the adductor magnus, adductor longus and adductor brevis. The accessory muscles are quadratus femoris, obturator externus, sartorius, pectineus, gracilis and the lower fibres of gluteus maximus. It is limited by contact with the medial aspect of the opposite limb, but a further range of movement is possible if the thigh is flexed. This further movement is limited, however, by the tension of the abductor muscles, the lateral band of the ilio-femoral ligament and the ligamentum teres. The position of equilibrium between abduction and adduction is obtained by the legs being slightly apart.

If the adductor muscles of the hip joint are paralysed, the patient cannot adduct his leg against resistance. There may also be weakness of flexion of the hip joint and lateral rotation of the thigh.

In bilateral paralysis, the late result is that the patient

gluteus maximus. The accessory muscles are the biceps femoris, semitendinosus, semimembranosus, the ischial fibres of the abductor magnus and the posterior fibres of the gluteus medius. It is limited by the tension of the flexor muscles of the hip joint and that of the ilio-femoral ligament.

Gluteus maximus is the main extensor of the thigh at the hip joint, and when the patient lies in the prone position there is weakness in raising the leg from the bed. The paresis or paralysis is, however, most noticeable when the patient becomes ambulant. There is also weakness of adduction and lateral rotation. If the upper fibres only are involved there may also be weakness of abduction and the initiation of medial rotation.

Later, when the patient puts his weight on the paralysed side, the body is thrown suddenly and forcibly backwards towards that side and the hip is hyperextended. There is a loss of the normal hyperextension thrust at the hip, and the patient quickly transfers his weight on to the normal limb. This results in an ungainly walk.

The patient complains of difficulty in attaining the erect position from a stooping or squatting position, and also in running or climbing stairs.

In bilateral cases, walking is extremely difficult, and the patient may require crutches.

Paralysis of the adductor magnus is observed by extension of the hip in outward rotation and the muscle acts on medial rotation. Extension can be carried out by either gluteus maximus or adductor magnus.

(3) *Abduction* : The range of movement is from 0° to 45° , and the principal muscles performing this movement are the gluteus medius and minimus. The accessory muscles are the upper fibres of the gluteus maximus, tensor fasciae latae, sartorius and piriformis. It is limited by the tension of the adductor muscles of the hip, by the medial band of the ilio-femoral ligament and by the pubo-femoral ligament.

Weakness of the abductors of the hip joint is observed when the patient is asked to raise his leg vertically from the bed, whilst in the "side lying" position. In paralysis of the gluteus medius, there is a weakness in abduction of the thigh,

- (a) *Tensor fasciae latae*—if this muscle is paralysed, genu varum may result and the leg is rotated laterally from the hip.
- (b) *Sartorius*—Paralysis of this muscle causes a weakness of flexion, abduction and lateral rotation of the thigh and of flexion of the knee
- (c) *Rectus femoris*—Paralysis of this muscle causes weakness of the extensors of the knee joint and also flexion of the hip joint.

(B) Movements of the Knee Joint

(1) *Flexion*. The range of movement is from 0° to 130° , and the principal muscles concerned are the biceps femoris, semitendinosus and semimembranosus. The accessory muscles are gracilis, sartorius and popliteus. It is limited by the contact of the calf with the posterior aspect of the thigh and by the tension of the ligamentum patellae and part of the posterior cruciate ligament.

If the patient's foot is on the ground, the gastrocnemius and the plantaris can assist in flexion of the leg.

If the patient has complete paralysis of the knee flexors, he is unable to flex the knee whilst lying in the prone position. If the biceps femoris, semitendinosus and semimembranosus are paralysed, there is a marked weakness in flexion of the knee joint, and a genu recurvatum is permitted by a hyperextension of the knee.

If the semitendinosus, semimembranosus and gracilis alone are involved, we get, in addition to the impaired flexion, an instability of the knee on its medial aspect, and this may be accompanied by a lateral rotation of the tibia on the femur. Later this can result in genu valgum. It should be noted, however, that hyperextension of the knee and lateral rotation of the tibia on the femur may also arise from paralysis of the popliteus muscle. If the biceps femoris alone is involved, there is an instability on the lateral side of the knee and possibly a medial rotation of the tibia on the femur. Later genu varum may arise.

Paralysis of the hamstrings may cause hyperextension, but

walks so that each footprint is directly in front of the previous one and he can only pass the rear foot forward by swinging his body round with each step.

(5) *Medial rotation* : The range of movement is from 0° to 45° , and the principal muscles concerned are tensor fasciae latae, anterior fibres of gluteus medius, and gluteus minimus. The accessory muscles are the ilio-psoas, pectineus and ischial fibres of adductor magnus. It is limited by the tension of the lateral rotator muscles, the ischio-femoral ligament and the posterior part of the capsular ligament. If the hip is extended, the tension of the ilio-femoral ligament also limits this movement.

If the medial rotators of the hip joint are paralysed, the leg is seen to lie in the bed in the position of lateral rotation. Normally internal rotation of the lower limb in the supine position is brought about by the effect of gravity, but weakness in the medial rotators may be present. Later the leg is seen to be externally rotated when the patient is standing or walking.

(6) *Lateral rotation* : The range of movement is from 0° to 45° and the principal muscles concerned are the quadratus femoris, obturator externus, obturator internus and the superior and inferior gemelli. The accessory muscles are the lower fibres of gluteus maximus, piriformis, adductor magnus, adductor longus, adductor brevis and the ilio-psoas. It is limited by the tension of the medial rotator muscles of the hip joint, and the lateral band of the ilio-femoral ligament.

If the lateral rotators of the hip joint are paralysed, the leg is seen to lie on the bed in a position of medial rotation and the thigh is adducted.

Weightbearing in late paralysis may result in genu valgum and lateral rotation of the lower leg. On the other hand, the patient may walk pigeon-toed. There is also a tendency for lumbar lordosis and an increased forward tilting of the pelvis.

(7) *Circumduction* : This is produced by a combination of the movements of flexion, extension, abduction and adduction.

There are, however, three muscles in the region of the hip joint which should be specially mentioned:

Another method frequently adopted by the patient is to place his hand on the knee or thigh and by pressing it backwards, he can prevent flexion of the knee joint

If the whole lower limb is medially or laterally rotated, it will be found to bear weight without flexing. The knee joint will also be found to lock if it is slightly hyperextended, but this position is definitely not advisable because in growing children it may lead to gross deformity

In cases with bilateral paralysis of the quadriceps femoris, the patient walks with his body bent well forwards and if talipes equinus is also present, the knee is extended and the weight is taken on the toes

(3) *Rotatory Movements* : Medial and lateral rotation are greatest when the knee joint is flexed at an angle of 90°

In medial rotation the principal muscles are the popliteus, semitendinosus and semimembranosus and the accessory muscles are sartorius and gracilis

This movement is checked by the tension of the biceps femoris muscle and by the anterior cruciate ligament

Paralysis of the popliteus and gracilis muscles results in a lateral rotation of the tibia

The only muscle concerned in lateral rotation is the biceps femoris, and it is checked by the tension of the medial rotators of the knee joint and the collateral ligaments

Paralysis of this muscle results in medial rotation of the tibia on the femur

Before leaving this section it is necessary to mention the action of two muscles.

(1) *Popliteus* —Paralysis of this muscle results in a weakness in flexion of the knee joint and in medial rotation of the leg at the beginning of flexion

(2) *Soleus* —If this muscle is paralysed in addition to the gastrocnemius, hyperextension of the knee is found to be a late result. This is more marked when the patient bears weight on the affected limb, and he finds it impossible to rise on to his toes. It is important, however, to note that a strong soleus muscle can compensate to a certain extent for

it must also be mentioned that hyperextension of the knee can be caused by a contracted tendo achilles. An extension strain is put on the knee by attempts to put the heel on the ground. If the biceps femoris alone is involved, there is instability of the lateral side of the knee.

In cases with unilateral paralysis of the hamstring muscles, rotation of the pelvis may occur, and in bilateral cases there may be lordosis of the lumbar spine.

If the patient is ambulant, hyperextension of the knee joint prevents a rhythmical progress in walking.

(2) *Extension.* The range of movement is from 130° to 0° , and the principal muscle concerned is the quadriceps femoris, especially the vastus medialis part, but the tensor fasciae latae may act as an accessory muscle. It is limited by the tension of the flexor muscles of the knee joint, the tension of the anterior cruciate ligament, the posterior fibres of the posterior cruciate ligament, the oblique posterior ligament and by the collateral ligament of the knee joint.

The quadriceps is a postural muscle and its main function is as a fixator of the knee joint, whilst maintaining the erect position. Paralysis results in a flexion deformity of the knee, but more often causes a backward displacement of the tibia on the femur thus producing a genu recurvatum. Trick extension can be produced by rectus femoris whilst the hip is extended.

If the quadriceps femoris muscle is paralysed, the patient is found to be unable to extend the knee, and if the hamstring muscles have been unaffected, a flexion contracture along with a contraction of the posterior capsule of the knee joint may result. Later this paralysis results in an instability of the knee joint, which becomes most evident when the patient is ascending or descending stairs.

In unilateral cases, where the hamstrings are not involved, the patient can walk without a caliper if he bends his body forwards and walks without fully extending the knee joint. It will therefore be necessary for him to thrust his weight forwards whilst the foot is held in the equinus position.

Walking may also be accomplished as the gluteus maximus can effectively extend the hip joint.

and the medial head of gastrocnemius, is limited by the tension of the peroneal muscle group, the lateral tarsal ligament and the contact of the tarsal bones medially.

Paralysis of tibialis posticus alone produces weakness of plantarflexion and of inversion of the foot. Eventually the patient is found to walk flatfooted as the longitudinal arch collapses, and talipes valgus is present. The patient also finds difficulty in rising on to his toes.

Paralysis of flexor hallucis longus produces weakness in inversion and a plantarflexion of the foot. Also, flexion of the interphalangeal and metatarsophalangeal joints of the great toes is weakened. Later there is pronation of the foot, a medial instability of the ankle joint, and the longitudinal arch is found to be less stable.

Flexor digitorum longus paralysis produces a weakness of plantarflexion, of inversion of the foot and of a flexion of the interphalangeal and metatarsophalangeal joints of the lateral four toes. Later the foot is found to be pronated and the longitudinal arch is weakened. The medial stability of the ankle joint is also impaired.

(2) *Dorsiflexion.* The principal muscle involved is the tibialis anticus and the accessory muscles are the extensor digitorum longus, extensor hallucis longus and peroneus tertius. This movement is limited by the tension of the tendo calcaneus and the posterior fibres of the deltoid and calcaneo-fibular ligaments.

Paralysis of the dorsiflexors of the foot with the sole exception of the tibialis anticus will produce inversion and dorsiflexion. This movement is limited by the tension of the peroneus longus and brevis muscles, the lateral tarsal ligament and the contact of the tarsal bones on the medial aspect.

In paralysis of the tibialis anticus alone, dorsiflexion and inversion of the foot is weakened. Later the forefoot is found to be abducted and the heel and tarsus everted. The longitudinal arch of the foot is also found to have collapsed. Paralysis of tibialis anticus is a very common finding in poliomyelitis and this is thought to be due to the area of cord representation being confined to a small zone.

If the extensor digitorum longus only is affected, there is

quadriceps paresis and this is done by pulling the leg back in passive knee extension.

(C) Movements of the Ankle Joint

(1) *Plantarflexion.* The principal muscles involved are the *gastrocnemius* and *soleus* and the accessory muscles are the *peroneus longus*, *peroneus brevis*, *plantaris*, *tibialis posticus*, *flexor hallucis longus* and *flexor digitorum longus*. This movement is limited by the tension of the extensor muscles, by the anterior fibres of the deltoid ligament and by the anterior talo-fibular ligament.

If the *gastrocnemius* is weak, there is a diminished plantarflexion of the foot, and flexion of the knee, but if it is completely paralysed there is dorsiflexion of the foot.

In unilateral cases, the patient will later develop a typical limp as he is unable to transfer his weight normally from the paralysed limb. In bilateral cases, he has an inelastic waddling gait and the feet are in the position of *talipes calcaneus* or *talipes calcaneo-cavus* deformity.

If the knee is flexed, plantarflexion at the ankle is carried out mainly by the *soleus* muscle, and if this is paralysed there is a weakness in plantarflexion.

Peroneus longus and *peroneus brevis* produce eversion of the foot from plantarflexion, and they are assisted in this movement by *extensor digitorum longus* and *peroneus tertius*. This movement is limited by the tension of the *tibialis anticus* and *posticus* muscles, the medial tarsal ligaments and the contact of the tarsal bones laterally.

In paralysis of *peroneus longus* and *brevis* muscles, the foot is held in the position of *talipes varus* or *talipes equinovarus*. There is also a lateral instability of the ankle joint and a weakness of the transverse and lateral longitudinal arches of the foot. Plantarflexion of the foot is also weakened. Later the patient is seen to walk on the lateral aspect of the foot, and to have difficulty in rising on to his toes.

Tibialis posticus is the main muscle producing foot inversion in plantarflexion. This movement which is usually assisted by *flexor digitorum longus*, *flexor hallucis longus*

digitorum is still active, the four lateral toes take up the position seen in cases with hammer toes.

- (4) Adductor hallucis—paralysis of this muscle allows medial displacement of the scaphoid and a hallux valgus deformity

As would be expected, the longitudinal and transverse arches of the foot may be weakened or obliterated by paralysis of the muscles and tendons helping to support them

(3) MUSCLES OF THE UPPER LIMB

The range of movement of the shoulder joint can be greatly increased if its movement is combined with that of the shoulder girdle. It is therefore advisable to discuss the scapular movements before proceeding to a detailed study of the movements of the shoulder joint.

(1) *Elevation of the Scapula.* This is brought about by the rhomboids, the upper fibres of the trapezius muscle and the levator scapulae, and it is limited by the tension of the costoclavicular ligament, the lower part of the capsule of the sternoclavicular joint and the tension of the pectoralis minor, subclavius and the lower fibres of the trapezius muscles.

(2) *Depression of the Scapula.* This is carried out by the force of gravity or by the action of the pectoralis minor, subclavius and the lower fibres of the serratus anterior. It is checked by the tension of the interclavicular and sternoclavicular ligaments and the antagonist muscles

(3) *Forward Movement of the Scapula Around the Chest Wall* This is carried out by the serratus anterior and pectoralis minor. It is limited by the tension of the antagonist muscles and the sternoclavicular ligament

(4) *Backward Movement of the Scapula* This is actively produced by the trapezius and rhomboid muscles. It is limited by the tension of the anterior sternoclavicular ligament and the anterior fibres of the costoclavicular ligament.

(5) *Forward Rotation of the Scapula* This is carried out by the trapezius and serratus anterior muscles

(6) *Backward Rotation of the Scapula* This is usually carried out by the influence of gravity, but it can be per-

weakness of dorsiflexion of the foot, but if this is combined with paralysis of *extensor digitorum brevis*, there is a weakness in extension of the interphalangeal and metatarsophalangeal joints of the toes. Later a talipes equino-varus deformity of the forefoot may develop.

Paralysis of *extensor hallucis longus* may produce a weakness of dorsiflexion of the foot and extension of the phalanges of the great toe. Later the great toe may be found to be plantarflexed and there is a tendency to dropfoot. If *extensor hallucis longus* is unaffected a noticeable deformity of a claw-shaped big toe is usually present due to its overaction.

Paralysis of the *peroneus tertius* alone produces a weakness of dorsiflexion and eversion of the foot, and later the foot is found to be inverted with a tendency to pes planus.

If all the dorsiflexors of the foot are paralysed, the foot is lifted higher than normal so that the toes may clear the ground. On completing the forward step, the toes will touch the ground first and are then followed by the heel.

(3) *Supination*. This takes place at the tarsal joints and the muscles which carry out the movement are the *tibialis anticus* and *posticus*. The accessory muscles which supinate the foot are *flexor hallucis longus* and *flexor digitorum longus*.

(4) *Pronation*. This takes place at the tarsal joints. The principal pronator is the *extensor digitorum longus* assisted by *peroneus longus* and *brevis*.

(D) Paralysis of the Muscles of the Foot

- (1) *Flexor hallucis brevis*—paralysis of this muscle produces a weakness of flexion of the metatarsophalangeal joint of the great toe. Later the great toe becomes hammer-toed and the longitudinal arch is weakened
- (2) *Flexor digitorum brevis*—as a result of paralysis of this muscle, there is a weakening of the longitudinal and transverse arches because of the loss of the muscular support
- (3) *Lumbricales*—as a result of paralysis of these muscles, the transverse arch is weakened and if the flexor

movement rapidly becomes weaker. If the trapezius is also involved, arm-raising exercises may be found to be impossible. Movements such as flexing the arm and pushing forwards of the arms also produce winging of the scapula.

- (d) **Trapezius** This is rarely completely paralysed although partial paralysis is often noticeable in portions of the trapezius. It controls the posture of the shoulders, can elevate and depress the scapula and fix the scapula during abduction of the arm.

If the trapezius muscle is paralysed, elevation and depression of the shoulder and rotation and adduction of the scapula are weakened or lost. Drooping of the shoulder may be very noticeable and extension of the head against resistance may also be impaired. Fatigue of the middle and lower fibres of the trapezius muscle often leads to a complaint of chronic backache.

The rhomboidius major and minor and levator scapulae are usually all involved together, and the scapula is then found to be abducted and the inferior angle rotated laterally. Adduction and extension of the arm is also noted to be weaker owing to the mobility of the scapula.

Rhomboid paralysis is noticeable by weakness of the downward rotators and adductors of the scapula. Winging of the vertebral border of the scapula is present at rest, but is more noticeable when the shoulder is braced. In testing the rhomboids, both upward and downward rotation must be tested. If both the trapezius and rhomboids are weak, it is necessary to carry out the test eliminating gravity.

(A) Movements of the Shoulder Joint

The scapulo-humeral movement takes place in an ordered sequence and therefore must be considered as a whole during muscle testing.

(1) *Flexion* The pectoralis major, coracobrachialis and the anterior fibres of the deltoid are the principal muscles concerned in this movement, but the biceps and subscapularis can act as accessory muscles. They are capable of raising the

formed actively by the levator scapulae and rhomboid muscles.

(7) *Scapular Abduction and Upward Rotation.* This is carried out by the serratus anterior acting as a prime mover and the pectoralis major as an accessory muscle. This movement is limited by the tension of the trapezius and rhomboid muscles.

(8) *Scapular Depression and Adduction.* This is carried out by the lower fibres of the trapezius muscle. It is limited by the tension of the upper fibres of the trapezius, the levator scapulae, the clavicular head of the sternomastoid and the interclavicular ligament.

(9) *Scapular Adduction and Downward Rotation.* The principal muscles are the rhomboidius major and minor, the trapezius muscle assisting in adduction. The movement is limited by the tension of the pectoralis major, pectoralis minor and serratus anterior muscles, and by the tension of the conoid ligament. Contact of the vertebral border of the scapula with the spinal muscles may also be a limiting factor.

Certain points should also be mentioned about the following muscles:

- (a) *Deltoid.* This muscle is more frequently affected than other muscles in this region, although paralysis in other adjacent muscles may often accompany the affected deltoid.
- (b) *Pectoralis minor.* Depression and extension of the shoulder is weakened in paralysis of this muscle. If the action of the respiratory muscles is impaired, involvement of pectoralis minor will further increase the difficulty of respiration.
- (c) *Serratus anterior.* Paralysis of this muscle produces weakness in protraction of the whole upper limb, and a winging of the scapula when the patient pushes his body away from a wall with his hands. The scapula lies a little higher and nearer to the midline and the trapezius contracts more strongly. Should he attempt repeatedly to raise the affected arm above his head, the

rotation to free the humerus from the acromion process. If the supraspinatus is completely paralysed, the patient is unable to abduct the shoulder joint through the first 10° , and the stability of this joint is also weakened.

The deltoid muscle consists of three parts—anterior, middle and posterior. In abduction, the anterior and posterior fibres act as synergists to neutralize the action of each other. Paralysis of the deltoid and supraspinatus can cause substitution in abduction by the biceps, serratus magnus and infraspinatus. If the deltoid is completely paralysed, abduction and medial and lateral rotation of the humerus are weakened. Subluxation downwards of the head of the humerus is a complication which must be guarded against. In very rare cases the patient may be able to abduct his arm through the first 90° , by contraction of the supraspinatus muscle.

If the upper fibres of the trapezius are involved, the patient may be unable to abduct the arm through the second 90° of abduction.

It is important always to test for abduction with the palms of the hands facing inwards, and both sides should be compared.

(4) *Adduction.* The two principal muscles concerned in this movement are the pectoralis major and the latissimus dorsi, but the coracobrachialis, subscapularis, infraspinatus, teres major, biceps and long head of the triceps may all be considered as accessory muscles.

(5) *Medial Rotation.* The range of movement is from 0° to 90° , and the muscles involved are the subscapularis, pectoralis major, teres major, latissimus dorsi and the anterior fibres of the deltoid. The movement is limited by the tension of the lateral rotator muscles of the shoulder and the superior portion of the capsular ligament.

In paralysis of the latissimus dorsi, extension, adduction, and medial rotation of the arm is weakened. The integrity of this muscle is most important in walking on crutches, in pulling the trunk upwards and forwards as in climbing and in swimming. Paralysis of this muscle will also interfere greatly with coughing and violent respiratory efforts.

arm to the horizontal position whilst serratus magnus rotates the scapula upwards. If the arm is in the fully extended position, the sternocostal part of the pectoralis major is the principal muscle in bringing the arm forward to the plane of the trunk.

If there is paralysis of the pectoralis major, there is a weakness of shoulder flexion, adduction and medial rotation. Later the patient may find that chopping and striking movements are difficult.

In paralysis of the coracobrachialis, flexion and adduction of the shoulder joint are found to be weakened.

If the upper fibres of the trapezius are involved, flexion of the humerus above the level of the shoulder may also be weakened.

(2) *Extension* The range of movement is from 0° to 50° . The posterior fibres of the deltoid and teres major are the principal muscles carrying out this movement, if it is commenced when the arm is by the side. To raise the arm backwards, the posterior fibres of the deltoid replace pectoralis major. If the fully flexed arm is extended against resistance, the latissimus dorsi, infraspinatus, teres minor, long head of the triceps, and the sternocostal head of the pectoralis major greatly assist this movement until the arm reaches the neutral position. It is limited by the tension of the flexor muscles of the shoulder joint and the greater tuberosity of the humerus with the acromial and coracoacromial ligaments.

Paralysis of the teres major produces a weakness in adduction, medial rotation and extension of the arm. In testing for extension of the humerus in the erect position, the acting muscles must be tested against gravity or resistance, because extension, depression and adduction of the humerus may be performed by gravity.

(3) *Abduction*. This takes place by a series of overlapping muscle actions. The first 10° of movement is initiated by the supraspinatus. The middle fibres of the deltoid and the supraspinatus raise the humerus to 75° . At 45° the movement is assisted by the serratus magnus until the arm reaches 75° . At this point, the scapula is rotated upwards and the arm is raised to 140° . The vertical position is achieved by outward

by pronator teres, and by the long flexors of the wrist and fingers with the forearm prone.

In considering the importance of muscles for function, the elbow flexors must have first place, because they are essential to the patient.

(2) *Extension* This is carried out by the triceps muscle and the muscles attached to the lateral epicondyle. It is limited by the tension of the flexor muscles of the forearm, the anterior radial and ulnar collateral ligaments of the elbow joint. It can also be limited by the olecranon process impinging on the olecranon fossa on the posterior aspect of the humerus.

In paralysis of the triceps, the extension of the elbow is greatly weakened and to a much less degree adduction. The patient is unable to push or throw whilst his arm is fully extended, and one of the greatest disabilities is that the patient may find it impossible to use crutches.

(C) Movements of the Radio-Ulnar Joints

(1) *Pronation* The range of movement is from 0° to 90° , and the pronator teres and pronator quadratus are the principal muscles. The palmaris longus, flexor carpi radialis and brachioradialis can act as accessory muscles. It is limited by the tension of the dorsal radio-ulnar, ulnar collateral and posterior radiocarpal ligaments. There may also be tension of the lowest fibres of the interosseus membrane. To test for pronation, place the arm at the side with the elbow flexed.

Paralysis of pronator teres produces a weakness in flexion of the elbow and in pronation of the forearm which usually lies in the supinated position.

(2) *Supination* The range of movement is from 0° to 90° , and the principal muscle is the supinator, but the biceps, brachioradialis, abductor pollicis longus and extensor pollicis longus may act as accessory muscles. This movement is limited by the tension of the pronator muscles of the forearm, of the oblique cord and the lowest fibres of the interosseus membrane. The anterior carpal ligament and the ulnar collateral ligament of the wrist joint also may be under tension.

Paralysis of the subscapularis produces a weakness of adduction and medial rotation of the arm.

(6) *Lateral Rotation.* The range of movement is from 0° to 90° , and the muscles concerned are the infraspinatus, teres major and the inferior fibres of the deltoid. This movement is limited by the tension of the medial rotator muscles of the shoulder, the superior portion of the capsular ligament and the coracohumeral ligament.

Paralysis of the infraspinatus produces a weakness of the lateral rotators of the arm, and if teres minor is also involved, there is an additional weakness in adduction. If teres minor and infraspinatus are both involved, the arm will be held in the position of medial rotation.

(7) *Circumduction.* This is produced by a combination of the movements of flexion, extension, abduction and adduction.

(B) Movements of the Elbow Joint

(1) *Flexion.* The range of movement is from 0° to 160° , and the muscles producing it are the biceps, brachialis, brachioradialis and those attached to the medial epicondyle. It is limited by the contact of the muscle groups on the flexor aspect of the arm, with those of the forearm, and by the contact of the coronoid process with the coronoid fossa of the humerus. It has also been said that flexion is limited chiefly by the tension of the structures on the posterior aspect of the joint.

If the biceps is paralysed, supination and flexion of the forearm is weakened. As the brachialis muscle is the chief flexor of the elbow joint, paralysis of this muscle produces marked weakness of this movement. If the brachioradialis is paralysed, the power of flexion of the elbow is slightly diminished and there is a weakness on supination and pronation of the forearm from the mid-position.

It is possible to detect weakness in each muscle by studying their separate actions. The biceps flexes in supination and pronator teres in pronation. When the biceps and brachialis are paralysed, local flexion of the elbow can be carried out

by pronator teres, and by the long flexors of the wrist and fingers with the forearm prone.

In considering the importance of muscles for function, the elbow flexors must have first place, because they are essential to the patient

(2) *Extension*. This is carried out by the triceps muscle and the muscles attached to the lateral epicondyle. It is limited by the tension of the flexor muscles of the forearm, the anterior radial and ulnar collateral ligaments of the elbow joint. It can also be limited by the olecranon process impinging on the olecranon fossa on the posterior aspect of the humerus

In paralysis of the triceps, the extension of the elbow is greatly weakened and to a much less degree adduction. The patient is unable to push or throw whilst his arm is fully extended, and one of the greatest disabilities is that the patient may find it impossible to use crutches.

(C) Movements of the Radio-Ulnar Joints

(1) *Pronation* The range of movement is from 0° to 90° , and the pronator teres and pronator quadratus are the principal muscles. The palmaris longus, flexor carpi radialis and brachioradialis can act as accessory muscles. It is limited by the tension of the dorsal radio-ulnar, ulnar collateral and posterior radiocarpal ligaments. There may also be tension of the lowest fibres of the interosseus membrane. To test for pronation, place the arm at the side with the elbow flexed

Paralysis of pronator teres produces a weakness in flexion of the elbow and in pronation of the forearm which usually lies in the supinated position.

(2) *Supination* The range of movement is from 0° to 90° , and the principal muscle is the supinator, but the biceps, brachioradialis, abductor pollicis longus and extensor pollicis longus may act as accessory muscles. This movement is limited by the tension of the pronator muscles of the forearm, of the oblique cord and the lowest fibres of the interosseus membrane. The anterior carpal ligament and the ulnar collateral ligament of the wrist joint also may be under tension.

The brachioradialis flexes the elbow in mid-position and does not act in pure supination or pronation.

If the supinator muscle is paralysed, there is weakness in supination of the forearm which usually lies in the pronated position. Supinator brevis can be tested with the elbow flexed.

(D) Movements of the Radiocarpal Joint

It should be noted that the range of movement of extension is greater than that of flexion, and these movements are limited chiefly by the tension of the antagonist muscles. Also, adduction is greater than abduction, and the movements are limited by the tension of the antagonist muscles and the lateral and medial ligaments of the joint respectively.

(1) *Flexion.* The principal muscles are the flexor carpi radialis, flexor carpi ulnaris and palmaris longus, whilst the flexor digitorum sublimis, flexor digitorum profundus and flexor pollicis longus can act as accessory muscles.

Paralysis of flexor carpi radialis produces a weakness of flexion of the wrist and elbow, and of the abductors of the hand. Pronation of the forearm may also be very slightly weakened.

If flexor carpi ulnaris is paralysed, there is a weakness on flexion of the elbow and wrist and on adduction of the hand.

If palmaris longus is paralysed, there is slight loss of power in flexion of the wrist and elbow, and also an inability to "cup" properly the palm of the hand.

Paralysis of flexor digitorum sublimis causes a weakness of flexion of the first interphalangeal joint of the fingers, the metacarpophalangeal joints, the wrist joint and to a much less extent, the elbow joint. The grip is also weakened. If the muscles are completely paralysed, the proximal interphalangeal joints may become hyperextended.

Paralysis of flexor digitorum profundus causes weakness of flexion of all the joints of the fingers and also weakness of wrist flexion.

When the flexor pollicis longus is paralysed, the patient finds difficulty in picking up small objects between the thumb

and fingers. Flexion of all the joints of the thumb is diminished and to a much less degree that of the wrist. In complete paralysis, there may be a hyperextension of the distal phalanx of the thumb.

(2) *Extension* : The principal muscles are the extensor carpi radialis longus, extensor carpi radialis brevis and extensor carpi ulnaris, but all the muscles whose tendons cross the posterior aspect of the wrist joint can act as accessory muscles

If there is paralysis of the extensor carpi radialis longus, extension and abduction of the hand at the wrist is weakened, and to a much less degree flexion of the elbow. If there is complete paralysis of extensor carpi radialis longus and brevis, adduction of the hand occurs.

If there is paralysis of extensor carpi radialis brevis alone, extension and abduction of the wrist is weakened and to a less extent, extension of the elbow.

Paresis of the extensor digitorum communis produces a weakness of extension of the fingers and of the wrist and elbow joints. If paralysis is complete, the fingers become flexed at the metacarpophalangeal joints.

If the extensor minimi digiti is paralysed, there is a weakness of extension of all joints of the fifth finger, and to a less extent extension of the wrist and elbow.

If there is a paresis of the extensor carpi ulnaris, extension of the wrist and elbow is weakened as well as adduction of the hand. If there is complete paralysis, the hand is seen to be abducted. To test the wrist extensors, the patient must try to grasp a ball. He can only do this with the wrist in slight extension because flexion of the fingers induces synergic contraction of the wrist extensors.

(3) *Abduction*. The muscles involved and which contract simultaneously are the flexor carpi radialis, extensor carpi radialis longus, extensor carpi radialis brevis, the abductor pollicis longus and the extensor pollicis brevis.

(4) *Adduction*. This is produced by the simultaneous contraction of the flexor carpi ulnaris and the extensor carpi ulnaris.

(5) *Circumduction*. This is produced by a combination

The brachioradialis flexes the elbow in mid-position and does not act in pure supination or pronation.

If the supinator muscle is paralysed, there is weakness in supination of the forearm which usually lies in the pronated position. Supinator brevis can be tested with the elbow flexed.

(D) Movements of the Radiocarpal Joint

It should be noted that the range of movement of extension is greater than that of flexion, and these movements are limited chiefly by the tension of the antagonist muscles. Also, adduction is greater than abduction, and the movements are limited by the tension of the antagonist muscles and the lateral and medial ligaments of the joint respectively.

(1) *Flexion.* The principal muscles are the flexor carpi radialis, flexor carpi ulnaris and palmaris longus, whilst the flexor digitorum sublimis, flexor digitorum profundus and flexor pollicis longus can act as accessory muscles.

Paralysis of flexor carpi radialis produces a weakness of flexion of the wrist and elbow, and of the abductors of the hand. Pronation of the forearm may also be very slightly weakened.

If flexor carpi ulnaris is paralysed, there is a weakness on flexion of the elbow and wrist and on adduction of the hand.

If palmaris longus is paralysed, there is slight loss of power in flexion of the wrist and elbow, and also an inability to "cup" properly the palm of the hand.

Paralysis of flexor digitorum sublimis causes a weakness of flexion of the first interphalangeal joint of the fingers, the metacarpophalangeal joints, the wrist joint and to a much less extent, the elbow joint. The grip is also weakened. If the muscles are completely paralysed, the proximal interphalangeal joints may become hyperextended.

Paralysis of flexor digitorum profundus causes weakness of flexion of all the joints of the fingers and also weakness of wrist flexion.

When the flexor pollicis longus is paralysed, the patient finds difficulty in picking up small objects between the thumb

on extension of all the joints of the thumb. To a less marked degree, supination of the forearm, extension of the wrist and abduction of the hand are also weakened. If the paralysis is complete, the distal phalanx is flexed.

Extension of the carpometacarpal joint and the metacarpophalangeal joint of the thumb is weakened if there is paralysis of extensor pollicis brevis. If there is complete paralysis, the proximal phalanx of the thumb is flexed.

(3) *Abduction of the Thumb at the Carpometacarpal Joint*
The range of movement is from 0° to 50° , and it is carried out by the abductor pollicis longus and brevis muscles. It is limited by the tension of the skin between the thumb and the index finger, and by the tension of the first interosseus muscle.

If the abductors are partially paralysed, the power of abduction of the thumb is greatly diminished and to a less degree, abduction of the thumb. If the paralysis of both muscles is complete, the first metacarpal is adducted as well as the whole hand.

(4) *Adduction of the Thumb at the Carpometacarpal Joint*
The range of movement is from 50° to 0° , and it is carried out by the adductor pollicis and the first palmar interosseus muscle. It is limited by the contact of the thumb with the lateral aspect of the second metacarpal bone.

If the adductor muscles are paralysed, the patient is unable to clench the thumb firmly over the closed fingers. Also the terminal phalanx of the thumb is fully flexed if a book is held between it and the index finger.

(5) *Circumduction* This is performed by combining the movements of flexion, extension, abduction and adduction.

(6) *Opposition of the Thumb to the Fifth Finger* This movement is carried out by the combined actions of the opponens pollicis and the opponens minimi digiti. It is limited by the tension of the extensor tendons of the first and fifth fingers and that of the transverse metacarpal ligament.

(F) Movements of the Metacarpophalangeal Joints

(1) *Flexion* The range of movement is from 0° to 70° , and in the thumb the principal muscles are the flexor pollicis

of flexion, extension, abduction and adduction movements of the wrist joint.

(6) *Flexion of the Radiocarpal Joint with Abduction.* This is produced by contraction of the flexor carpi radialis muscle. It is limited by contact of the trapezium with the styloid process of the radius, and by the tension of the posterior radiocarpal and ulnar collateral ligaments.

(7) *Flexion of the Radiocarpal Joint with Adduction.* This is produced by the contraction of the flexor carpi ulnaris. It is limited by the tension of the posterior radiocarpal and radial collateral ligaments.

(8) *Extension of the Radiocarpal Joint with Abduction.* This is due to the combined action of the extensor carpi radialis longus and the extensor carpi radialis brevis. It is limited by the contact of the trapezium with the styloid process of the radius and by the tension of the anterior radiocarpal and ulnar collateral ligaments.

(9) *Extension of the Radiocarpal Joint With Adduction.* This is produced by the action of the extensor carpi ulnaris. It is limited by the tension of the anterior radiocarpal and radial collateral ligaments.

(E) Movements of the Thumb

(1) *Flexion and Opposition of the Thumb at the Carpometacarpal Joint.* These movements are produced by the action of the flexor pollicis longus, flexor pollicis brevis and the opponens pollicis. The adductor pollicis may act as an accessory muscle.

Paralysis of flexor pollicis brevis produces a hyperextension of the proximal phalanx.

If there is paralysis of the opponens pollicis, there is a flattening of the thenar eminence and a weakness of the grip between the fingers and the thumb.

(2) *Extension of the Thumb at the Carpometacarpal Joint.* This movement is produced by the extensor pollicis longus and the extensor pollicis brevis, and the abductor pollicis longus may act as an accessory muscle.

Paralysis of extensor pollicis longus produces a weakness

(G) Movements of the Interphalangeal Joints

(1) *Flexion* The range of movement is from 0° to 90° . In the case of the thumb, this is carried out by the flexor pollicis longus, and for the fingers by the flexor digitorum sublimis and the flexor digitorum profundus. The latter muscle, however, acts only on the distal joints. The movement is limited by the tension of the expansions of the extensor digitorum communis tendons.

(2) *Extension*. The range of movement is from 90° to 0° . In the case of the thumb, extensor pollicis longus carries out this movement, and in the case of the fingers, the lumbricales, palmar interossei, dorsal interossei and extensor digitorum communis are the muscles concerned.

Special points should be noted about the following muscles:

- (a) Opponens and flexor minimi digiti—paralysis of these muscles produces a flattening of the hypothenar eminence and the fifth finger cannot be approximated to the thumb.
- (b) Lumbricales—paralysis of these muscles produces a hyperextension of the metacarpophalangeal joints and a flexion of the interphalangeal joints.
- (c) Palmar interossei—paralysis of these muscles causes the patient to have difficulty in adducting the index, ring and little fingers. Flexion of the metacarpophalangeal joints and extension of the interphalangeal joints are also weakened.
- (d) Dorsal interossei—paralysis of these muscles causes the patient to have difficulty in abducting the index, middle and ring fingers. The metacarpophalangeal and interphalangeal joints are affected in the same manner as in paralysis of the palmar interossei.

longus and the flexor pollicis brevis. They are assisted by the first palmar interosseus muscle, and this movement is limited by the tension of the tendons of the extensor muscles of the thumb.

The range of movement of the flexors of the fingers at these joints is from 0° to 90° . In the fingers the principal muscles are the flexor digitorum sublimis and the flexor digitorum profundus. The accessory muscles are the lumbricales and interossei, and the little finger has in addition the flexor minimi digiti. This movement is limited by the tension of the expansions of the extensor tendons of the fingers.

(2) *Extension*. The range of movement is from 70° to 0° , and in the thumb the only muscles concerned are the extensor pollicis longus and extensor pollicis brevis. It is limited by the tension of the anterior and collateral ligaments of the joint.

The range of movement in extension of the fingers is from 0° to 30° , and the principal muscle is the extensor digitorum communis. This is assisted by the extensor indicis and the extensor minimi digiti in the case of the index and the little fingers respectively. It is limited by the tension of the flexor muscles of the fingers and of the anterior and collateral ligaments.

If the extensor indicis is paralysed, the power to extend the index finger is diminished and to a less marked degree, extension and abduction of the wrist joint.

(3) *Abduction*. The range of movement is from 0° to 25° , and the principal muscles concerned are the dorsal interossei. The accessory muscles are the long extensors of the fingers, and this movement is limited by the tension of the fascia and skin between the fingers.

The little finger has a common muscle of its own, namely, the extensor digiti minimi, which is inserted into the base of the fifth metacarpal bone.

(4) *Adduction*. The range of movement is from 25° to 0° , and it is carried out by the palmar interossei. It is limited by the fingers coming in contact.

tion of the cerebrospinal fluid shows a lymphocytic pleocytosis with an increase in the protein content, but there is also a reduction in the chlorides and sugar content. Jaccottet (1933) stated that a cell count below 300 per cmm., a lymphocytosis, normal sugar and chloride levels are characteristics of poliomyelitis. An X-ray of the chest and a Mantoux test are also advisable.

(2) *Pyogenic Meningitis*. In this condition, marked pyrexia and rigors may occur, and the lungs, ears and mastoids should be examined carefully. Irrationality and coma are more common in this condition than in poliomyelitis.

The cerebrospinal fluid is cloudy and polymorphonuclear leucocytes are present, the total number ranging from 100 to 1,000 per cmm. Organisms such as meningococci, staphylococci, streptococci and pneumococci may be isolated on culture, and the sugar content is diminished or absent.

(3) *Cerebral Abscess*. This may also be secondary to disease of the lungs, ears and mastoids, but the cerebrospinal fluid may be quite normal.

(4) *Post-infective Encephalitis*. Here there is usually a history of a recent attack of measles, mumps, chicken pox or vaccination against smallpox. Disorientation or convulsions with spasticity of the extremities is usually present and the sugar content of the cerebrospinal fluid is invariably increased. Differential diagnosis may not be possible on clinical grounds alone unless typical segmental lower motor neurone paralysis is also present.

(5) *Toxic Encephalitis*. This is usually encountered as a complication of pneumonia, dysentery or typhoid.

(6) *Acute Encephalomyelitis*. This may be a complication of an inoculation of the rabies vaccine.

(7) *Lymphocytic Choriomeningitis*. This is only diagnosed by the isolation of the virus from the cerebrospinal fluid or blood of the patient. Diagnosis is also assisted by demonstrating a rising total of antibody in the blood.

(8) *Subarachnoid Haemorrhage*. Signs of meningeal irritation may be marked and the cerebrospinal fluid is usually blood-stained.

(9) *Acute Aseptic Lymphocytic Meningitis*. This is a rare

CHAPTER IX

DIFFERENTIAL DIAGNOSIS

IN the initial stages, this may be extremely difficult, because up to the present time no specific clinical or laboratory test has been found. The observer must therefore be thoroughly familiar with all the early clinical signs and symptoms and always on the alert to detect any new features which arise.

Early diagnosis is important for two particular reasons. First of all, it enables adequate isolation to be carried out and so prevent further spread of the disease, and in the second place, adequate treatment can be carried out by the poliomyelitis team.

The following classification, although admittedly incomplete, is given in an attempt to clarify this very difficult problem.

(I) Abortive and Preparalytic Stages

Here the general systemic disturbances predominate and a differential diagnosis from acute nasopharyngitis, laryngitis, acute follicular tonsillitis, acute otitis media, and acute sinusitis must be considered.

During an epidemic, any patient with influenzal symptoms or a feverish chill must be kept under careful observation, and if cervical lymphadenitis is present, peritonsillar abscess, diphtheria, lymphatic leukaemia, and the acute stages of scarlet fever must be borne in mind.

(II) Meningeal Irritation

Marked meningeal reactions at the onset of the prolonged preparalytic stage have led to a diagnosis of meningitis or meningoencephalitis. If the patient shows evidence of meningeal irritation, a careful clinical examination must be made to eliminate the possibility of the following.

(1) *Tuberculous Meningitis* This is usually suspected by the insidious onset and the slow progressive course. Examina-

Petechiae may appear on the skin, and ecchymoses in the region of the joints may interfere with their movements. The cerebrospinal fluid is normal and the radiographic appearance of the bones is typical.

(8) *Osteomyelitis* This may arise from trauma or as a result of septicaemic or pyaemic infection. It usually commences abruptly with a rigor followed by a high temperature and severe pain in the limb which may become swollen, oedematous and congested. The maximum point of tenderness is usually situated over the bony focus.

(9) *Synovitis* If traumatic, there is an effusion of blood and synovial fluid into the joint cavity and pain on the least movement which stretches the injured part.

If due to typhoid, it is a blood-borne infection and may occur in several joints, but if pyococci are present, the symptoms are those of a suppurative arthritis.

(10) *Tuberculosis of Bone or Joint*. This is secondary to some tuberculous focus in the body, and radiographs are helpful only in the later stages. The onset is insidious, with slight impairment of the movement of the joint and pain in the leg. The pain in the bone is worse after exercise, and a slight lump develops with wasting of the local muscles. In the later stages, an abscess may form.

(11) *Polyneuritis* The onset is gradual and sensory changes are frequently detected in the distal parts of the limb. The distribution is symmetrical and the legs are usually more severely affected than the arms. The distal groups of muscles are more severely affected than the proximal groups, and the muscles are tender on pressure. The tendon reflexes are lost but the cutaneous reflexes are retained.

(12) *Peripheral Neuritis (Diphtheritic)* Faucial diphtheria is frequently complicated by paralysis of the palate and occurs from the fourth to the sixth week. Multiple neuritis may also occur about the fifth week after recovery from the throat infection. There is weakness and aching of the limbs and unsteadiness in walking. Sensory ataxia is almost always present and it is often severe when the paralysis is trivial.

(13) *Acute Transverse Myelitis*. The important points to

condition involving the central nervous system, but it should nevertheless be considered.

(10) *Trauma*. In this case, the history and clinical examination should establish the diagnosis.

(III) Conditions other than Poliomyelitis producing Paralysis or Pseudoparalysis

(1) *Acute Rheumatic Fever*. Here there is an absence of neck and back rigidity, and pain is associated with movement of the joint. If the extremity is relaxed, there is no real muscle tenderness, but if an attempt to move the joint is made, the muscles around are voluntarily contracted. The reflexes are unimpaired and the cerebrospinal fluid is normal. Signs in the heart also aid the diagnosis.

(2) *Erythema Nodosum*. In this disease, the local lesions are bilateral and occur chiefly over the legs. These lesions are round, deep red or purple tender swellings on the extensor aspects of the limb.

(3) *Suppurative Arthritis*. This may accompany septicaemia or pyaemia. It is usually acute and there is a great deal of pain, swelling and redness around the joint. If the joint is aspirated, the offending organism can usually be obtained from the joint effusion.

(4) *Traumatic Arthritis*. A history of trauma is always obtained and blood may be aspirated from the joint.

(5) *Epiphysitis* : (a) Traumatic; (b) non-traumatic, e.g. syphilitic.

This is associated with other syphilitic signs, by pain on movement, and contraction of the muscles when the skin is irritated.

(6) *Rickets*. The points to be noticed here are the presence of a rachitic rosary, delayed dentition, and an open anterior fontanelle. A radiograph is conclusive as it shows the typical cupping of the diaphysis, the irregular and ill-defined epiphyseal line and the poorly ossified epiphysis.

(7) *Scurvy*. In addition to the obvious malnutrition and bleeding gums, the spasticity of the muscles and resistance to movement because of the ensuing pain, are evident.

Petechiae may appear on the skin, and ecchymoses in the region of the joints may interfere with their movements. The cerebrospinal fluid is normal and the radiographic appearance of the bones is typical.

(8) *Osteomyelitis*. This may arise from trauma or as a result of septicaemic or pyaemic infection. It usually commences abruptly with a rigor followed by a high temperature and severe pain in the limb which may become swollen, oedematous and congested. The maximum point of tenderness is usually situated over the bony focus.

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(12) *Peripheral Neuritis (Diphtheritic)*. Faucial diphtheria is frequently complicated by paralysis of the palate and occurs from the fourth to the sixth week. Multiple neuritis may also occur about the fifth week after recovery from the throat infection. There is weakness and aching of the limbs and unsteadiness in walking. Sensory ataxia is almost always present and it is often severe when the paralysis is trivial.

(13) *Acute Transverse Myelitis*. The important points to

be noted are that above the lesion there are no disturbances of sensation, movement or reflexes, but at a point corresponding to the upper margin of the lesion there is a zone of flaccid hyperaesthesia. Below the lesion there is complete paralysis of both legs, loss of sensation and loss of sphincter control. Spasticity and increased reflexes occur later and are accompanied by a spastic gait. Slow atrophy of the affected muscles and progressive paralysis are also seen.

(14) *Fractures and Dislocations* A recent history of trauma is always obtainable and these should cause no difficulty.

(15) *Trichinosis*. In this disease, the patient complains of severe muscular pain. There is a history of having eaten uncooked pork, and examination of the blood shows a definite eosinophilia.

(16) *Muscular Dystrophies*. There is either a marked hereditary influence or a family predisposition, as the disease is known to affect several members of the same generation. All the different types begin in childhood or round about puberty, and the muscles of the trunk and proximal parts of the limbs are the ones most severely affected. The distal parts of the limbs invariably escape and fibrillary tremors are absent. The electrical reactions are found to be only quantitatively diminished.

(17) *Myasthenia Gravis*. In this disease the muscles of the eyes, lips and tongue are readily exhausted, but soon recover their pre-exercise power on resting. Later the muscles of the limbs and those of respiration are affected. Paraesthesia is occasionally present.

(18) *Friedrich's Ataxia*. This usually occurs in several brothers and sisters about the age of puberty. The speech is affected and nystagmus is present. There is inco-ordination of a jerky staggering nature which affects first the legs and later the trunk. Scoliosis and kyphosis may develop.

(19) *Tumours of the Spinal Cord*. If these are within the cord, there occurs a slowly developing paralysis and anaesthesia which may be primarily unilateral. Later rigid paralysis and exaggerated reflexes are present. If the meninges or vertebral column are the seat of the tumour,

pain may be the first symptom, and this is due to the irritation of the nerve roots.

(20) *Cerebral Vascular Lesions* : These are uncommon in young subjects, but should always be borne in mind. In cerebral thrombosis there may be cyanosis of the face, oedema of the eyelids and face, and protrusion of the eyes.

(21) *Spastic Paralysis and Congenital Deformities* : Spastic paraplegia, spina bifida, congenital club foot, congenital dislocation of the hip, etc., are the commonest examples.

(22) *Progressive Muscular Atrophy*. A gradually increasing weakness and disability commences about puberty. There is atrophy of the muscles from the lower motor neuron lesion,

anaesthesia, muscle atrophy and trophic changes usually affecting males.

Should these last three occur (21-23), they will cause difficulty in diagnosis only in the late stages of the disease.

(IV) Other Conditions

These must be included for completeness, although they should give rise to very little difficulty.

(1) *Acute Appendicitis* : A careful abdominal examination and a white cell count should determine the diagnosis.

Holzer (1922) reported on a case with the simultaneous occurrence of adult poliomyelitis and perforated appendix.

(2) *Acute Pyelitis* : An examination of the urine will usually indicate the diagnosis.

(3) *Guillane-Barré Syndrome (Radiculoneuritis)* : There is no stiffness of the neck and the weakness or paralysis tends to spread. There is a high protein content in the cerebrospinal fluid but the number of cells is normal.

(4) *Typhoid and Paratyphoid Fever* : This can be diagnosed conclusively by the positive Widal reaction.

(5) *Undulant Fever* : The insidious onset and characteristic temperature chart should be noted. There is

enlargement of the liver and spleen and neuralgic pains are common. Culture of the blood may show the organism. The agglutination reaction also develops

- (6) *Malaria* : Examination of the blood film will establish the diagnosis
- (7) *Dysentery* : There is colicky abdominal pain and frequent small dysenteric stools. Marked dehydration may be present and the patient may become delirious.
- (8) *Colitis*. There may be frequent fluid offensive stools and abdominal colic
- (9) *Debility After a Febrile Illness*. The history should be carefully considered, after which there should be no difficulty in diagnosis.
- (10) *Hysterical Paralysis* : This should present no difficulty to an alert clinician. Young's cases (1947) were differentiated by
 - (a) The diffuse nature of the weakness with normal or increased muscle tone and reflexes.
 - (b) Bizarre sensory changes not corresponding to any anatomical distribution.
 - (c) Normal cerebrospinal fluid.
 - (d) Neurotic features in past and present history.

BIBLIOGRAPHY

- HOLZER, P (1922) *Med Klin*, 18, 110
JACCOTTET, M (1933) *Arch Méd Enf*, 36, 528
YOUNG, H (1947) *New Engl J Med*, 236, 794

CHAPTER X

TREATMENT

THIS is a most difficult problem, because the literature abounds in articles by competent observers most of whom claim that their specific form of treatment undoubtedly gives the best results. If these articles are studied in detail, it will be observed that in the majority, the underlying principles are the same, but the method of their application varies. These claims are most difficult to assess because the disease varies in intensity from epidemic to epidemic, and the number and severity of the paralytic cases varies accordingly. Also, most observers attempt to fit each individual case into a certain specific treatment group, whereas in our opinion, each patient must be dealt with as an individual problem in order to obtain the best results.

Throughout the whole of the treatment, it is absolutely essential that all the members of the poliomyelitis team should remember that the condition of the patient is constantly changing in character. Whatever method of treatment has been decided upon, it must be repeatedly reviewed, so that it can be adapted or changed as the condition of the patient alters with his response to treatment. These alterations should always be carried out gradually and with the greatest care, and children especially must be supervised during the growing period, otherwise deformities may progress, sometimes with great rapidity.

The treatment should vary according to the age of the patient, the stage of the disease and the time which has elapsed between the onset of the disease and the commencement of adequately supervised treatment.

If cases are seen in the later stages, it is essential to know the extent of the original involvement and then to ascertain the extent of the paralysis, paresis or deformities present before considering what improvement there has been in the individual muscles. The type of previous treatment should also be ascertained, and then having considered all the

relevant factors, a definite line of treatment should be embarked upon.

It has been noticed that poliomyelitis is primarily a disease of the nervous tissue and secondary changes are seen in the muscles, fascia and skin, either as a direct result of nerve impulse, loss of motion or interference with circulation. It is therefore unreasonable to suppose that the actual nerve paralysis can be diminished by the early and constant treatment of the muscle.

Let us now give a résumé of the essential points in the treatment of a case of poliomyelitis.

- (1) Absolute mental and physical rest during the acute stage, preferably in a darkened room.
- (2) Skilled nursing technique
- (3) Careful diagnostic study.
- (4) The maintenance of correct body alignment of the affected parts, and checking at frequent intervals to prevent or limit any tendency to stretching or contracture
- (5) Light casts or splints carefully applied when indicated, *especially for night use in children to prevent stretching of the weakened muscles.*
- (6) Warmth to promote the circulation and thus improve the nutrition, and elimination of waste products from the paralysed parts.
- (7) Physiotherapeutic treatment to prevent stiffness of the joints and to counteract the effects of muscle spasm
- (8) Accurate localized muscle re-education, as soon as active motion is possible without pain or muscle irritation.
- (9) The avoidance of weight-bearing during the period of recovery.
- (10) The restoration of maximum function, joint stability, and the maintenance of correct posture
- (11) Constructive psychotherapy by encouragement and reassurance.
- (12) Over-treatment must be guarded against, and adequate periods of rest enforced, as gross muscular fatigue must be avoided.

Recovery passes from stage to stage, being helped by rest and relaxation, but it is hindered by overstrain which may turn a partial paralysis into a complete one

In the abortive case, the patient should be confined to bed in his home for two or three days after the temperature has returned to normal. In the preparalytic case, the patient should be at complete rest in bed for at least four weeks, and in cases which have shown marked initial symptoms, for a period of not less than six weeks.

In the preparalytic stage, the doctor may be uncertain whether he should transfer the patient to hospital immediately, and until this has been decided, the patient must be kept very quiet both physically and mentally by adequate doses of sedative drugs so as to prevent restlessness and to promote sleep. At the first stage of bulbar palsy, hospital treatment is urgently required because of the danger of a rapid spread to the respiratory centre.

For prophylaxis of known or suspected infections, a normally practised aseptic technique is most important. The suspected case may be suffering from meningitis, rheumatic fever, etc., and an efficient fever hospital is the best place to treat cases of this kind. If the case is one of poliomyelitis, barrier nursing for approximately three weeks is required, and it is therefore best to admit the case to an isolation hospital. Here the nursing staff have the necessary experience in the use of the respirator and in the nursing of tracheotomy cases.

Gauvain and Langston (1939) advised that all cases should be admitted to a special ward of an orthopaedic hospital immediately on diagnosis and not to a medical ward of a general or isolation hospital. On the other hand, a general hospital may be considered to be the best place as it offers all the general and special medical and nursing services. Special hospitals, certainly in this country, are impractical and uneconomical from the medical, nursing and financial points of view. The main reason for this is that the disease is a seasonal one, and therefore the facilities would be out of use for long periods of time.

It is, however, most essential to have a full team consisting

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It can further be sub-divided into the acute febrile stage and the early convalescent stage, the latter commencing whenever the temperature has remained within normal limits for twenty-four hours, even although the muscles are still very tender and irritable.

During this stage, an attempt is made by rest and heat to localize and arrest the infection. The relief of pain and the prevention of joint stiffness and deformity are also necessary.

The Second Stage

This is the intermediate stage and commences when the tenderness of the muscles disappears. It varies considerably in duration and the treatment is directed mainly to assist the recovery of the paretic and paralysed muscles.

The Third Stage

This is the residual stage and is usually reached about two years from the onset of the disease. By this time, the surgeon will usually have decided upon the advisability of the prolonged or permanent use of appliances or of operation.

ACUTE STAGE

The acute febrile stage usually lasts for four to seven days and occurs during the three-week period of isolation in the fever hospital. During the acute stage, expert nursing combined with rest and relaxation of the muscles are essential. The patient is not allowed to sit up, and passive movements, active exercises, etc., are contra-indicated.

All cases should be seen by the orthopaedic surgeon within twenty-four hours of the onset, and a muscle chart may be made out at this time to record the power of the affected muscles. If the patient complains of severe pain or is very apprehensive, it is better to postpone muscle testing for a day or two, bearing in mind that the earliest possible estimation of the paralysis is advisable. Some orthopaedic surgeons do not make a full muscle chart at this stage if the patient is in the neutral position, as they consider that it is unneces-

of a physician, orthopaedic surgeon, physical medicine specialist, physiotherapist, occupational therapist, almoner and a fully trained nursing staff, all of whom combine their own special knowledge for the ultimate benefit of the patient.

In the paralytic cases during the first four to six weeks, the clinician is able to get a complete picture of the functional weakness. The proposed treatment may be conveniently divided into three stages, but we must remember that this division is not a rigid one but that one stage invariably merges into another. Although the different forms of treatment are discussed under separate headings, each particular method may be preferred to a greater or lesser extent by one observer than by another during the first and second stages.

The First Stage

The acute stage usually lasts about three weeks, but rarely it may be prolonged over several months, as it extends from the time of onset until the disappearance of muscle pain and tenderness.



FIG 8—NOTE POSITION OF BODY AND LOWER LIMBS
(Reproduced by permission of the Nursing Mirror)

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is in the neutral position, as they consider that it is unneces-

sary to have more than a rough idea of the muscular paralysis until the treatment is being planned.

At least 30% of all cases fail to develop detectable paralysis or develop a temporary paralysis which disappears in seven to fourteen days. In these cases, no specific treatment is indicated, as recovery depends upon the spontaneous return of the function of the anterior horn cells which have not been destroyed. The effects of pain and muscle spasm should be mitigated, but interference must be minimal whilst the infection is active.

Nuchal rigidity and stiffness of the back usually disappear spontaneously with the departure of the meningeal irritation. Intravenous injection of 100 to 250 cc of 25% glucose solution every twelve to twenty-four hours and lumbar puncture, might diminish pain, rigidity and headache. The lumbar puncture should be repeated only when symptoms of increased cerebral pressure are manifest, and can then be repeated daily until the pressure is normal.

All cases, especially with spinal and abdominal muscle paralysis, should be nursed on a firm mattress which can be made more rigid by the use of fracture boards, but a plaster bed or Bradford frame and restraining harness may be required for a restless child. The maintenance of the correct posture is essential whether the patient is lying in the lateral, prone or supine position, and this is especially important during sleep.

When palatal paralysis only is present, the patient should be given semi-solid or soft foods, but if the muscles of deglutition are involved, he is unable to swallow saliva, mucus or vomitus. There is then a mechanical blockage of the upper respiratory passages and usually small regions of atelectasis occur. In these cases, the mechanical respirator is contra-indicated, unless a previous tracheotomy has been performed, otherwise the contents of the throat may be sucked into the lungs producing an aspiration pneumonia. The correct treatment is postural drainage as advised by Brahdry and Lenarsky (1934) and Stimson (1940). The patient lying in the prone position may be tipped 20° to 30° from the horizontal and a suction apparatus used with care and

skill is useful in removing the mucus and saliva. Certain observers advise intranasal feeding with milk, glucose water, eggs, etc., but it must be used with the greatest caution otherwise collapse and death may follow the feeds. We prefer to give 5%-10% intravenous glucose solution in the beginning, and if the patient vomits gastric suction should be commenced.

Atelectasis of the lungs may be treated by immediate bronchoscopy, and aspiration by suction as advocated by Morrow and Stimson (1947).

Harwood Stevenson (1952) recommended the following treatment for the management of respiratory infection in patients who were severely handicapped by poliomyelitis.

The chest should be X-rayed at regular intervals and a note made of the position of the diaphragm during inspiration and expiration. The vital capacity should also be recorded. Throat or nose infection should be reported at once and a course of chemotherapy given. Swabs should also be taken so that the sensitivity tests of the organism are known.

Before there is any evidence of blockage, postural drainage and assisted coughing should be given. Any asymmetrical movement or deficient air entry should be noted. This is most important as it is well known that the presence of air beyond the block greatly aids the assisted coughing and helps to dislodge the mucus plug. The position of the patient also aids drainage towards the tracheal bifurcation, but the foot of the bed should not be raised more than 20°. If it is elevated more than this, we may get even greater distress owing to the pressure of the abdominal viscera on the weakened diaphragm.

Assisted coughing is the name given to sudden firm bimanual pressure on the chest which is synchronous with the patient's feeble coughing. It is kept up for twenty minutes at a time and is repeated four times a day.

Galloway (1943) pointed out that tracheotomy may be a life-saving measure in selected cases. Kubecek *et al.* (1948) stated that if laryngeal and pharyngeal paralysis are observed along with an elevated temperature and a fast pulse, then tracheotomy should be performed and oxygen therapy at a

slightly positive pressure commenced to prevent pulmonary oedema and anoxia of the already infected nerve cells.

The main indications for tracheotomy are:

- (1) Irregular shallow breathing and/or periodically apnoeic breathing
- (2) Agitation, extreme restlessness and apprehension preventing pharyngeal exploration.
- (3) Progression of bulbar involvement with increasing dyspnoea.
- (4) Presence of congestion and recurrent cyanosis.
- (5) Coarse râles in the chest and laryngeal stridor.
- (6) Stupor or exhaustion in which the patient is apparently unaware of the accumulation of secretions in the pharynx.
- (7) Inability to cough effectively
- (8) Paralysis of the vocal cords (Bilateral abductor paralysis).
- (9) Intralaryngeal hyperaesthesia which has been demonstrated by laryngoscopy.

In spite of all the foregoing indications, it must be pointed out that no definite rule can be given as to when a tracheotomy should or should not be performed. All the clinical features in each particular case should be taken into account before arriving at a definite decision. It should, however, be performed preferably before cyanosis occurs, because if this sign is waited for, irreversible damage may have been done to an already virus-damaged central nervous system.

For immobilization in the acute stage, the following positions of election are advised:

Spine As the patient is invariably nursed in the recumbent position on a firm mattress, a Whitman or a Bradford frame, the bed itself acts as a most efficient splint and additional apparatus to maintain the desired positions of the spine is rarely required. The normal curves are thus maintained and flexion and lordosis of the lumbar spine avoided. Simple restrainers can be used to prevent a child sitting up.

Hip In children, the legs should be abducted so that the angle between them measures 30° , but in adults the joint is abducted to 15° to 20° with flexion varying from 0° to 20° .

Care is taken to see that there is no rotation of the limb. Some observers advise that in adults the paralysed legs should be kept extended, adducted and internally rotated. I personally do not advise internal rotation because the foot will then naturally fall into a varus position and I consider that the mid position of the limbs is the best.

Knee. In order to avoid stretching of the quadriceps muscle and posterior capsule of the joint with a resulting hyperextension and instability, the knee is flexed 5° to 15° over a pillow, and care is taken to prevent varus and valgus deformity.

Ankle. In cases where there is a tendency to drop-foot, the foot is placed at right angles to the tibia without inversion or eversion. A foot-rest covered with a pillow should be placed against the feet to keep them in this position and a bed-cradle used to avoid pressure by the bedclothes.

Tarsal Joints. The foot is placed in the neutral position so that there is no inversion or eversion.

Toes. Every effort should be made to maintain flexion of the metatarsophalangeal joints and the extension of the interphalangeal joints as this helps to maintain the activity of the intrinsic muscles of the sole.

Shoulder. If the abductors of the shoulder are involved, the shoulder should be abducted to 75° without rotation, otherwise abduction to 45° with flexion of 30° and external rotation of 15° will be sufficient to prevent contraction of the adductor muscles, capsular stretching and subluxation of the joint.

Elbow. If there is paralysis of the muscles of the elbow, the joint should be maintained in flexion between 60° and 90° .

Forearm. The position should vary according to the muscle weakness, but it is usually one midway between supination and pronation. Pronation, which is more useful than supination, can be obtained more easily by gravity.

Wrist. Extensor paralysis usually predominates and the wrist should be placed in 30° of extension without deviation. This angle should be measured along the dorsum of the radius and the third metacarpal. A simple cock-up splint which does not extend beyond the necks of the metacarpals is advisable.

for this purpose. The fingers should be relaxed so that the flexor muscles can be used.

Digits and Thumb. The hand is placed in a position similar to that in holding a tumbler. The thumb is in the line of the anterolateral border of the radius and all the joints of the fingers are flexed to about 25°.

Overstretching of the paralysed thenar muscles should be prevented by the application of a small plaster of Paris splint, having its broad end lying over the lateral surface of the distal end of the radius and its narrow end curved round into the web between the thumb and index finger.

For paralysis of the intrinsic muscles of the hand, a bar of plaster of Paris is placed across the palm at the level of the necks of the metacarpals, and this is joined by two transverse dorsal bars lying parallel across the metacarpals and proximal to the phalanges. The knuckles protrude between them. The metacarpophalangeal joints are thus held in a position of flexion of 45°, as extension of these joints must be avoided.

The positions for immobilization just mentioned are intended to act as a guide to the treatment of the muscle groups and joints actually affected, and they should not be used prophylactically in every case. Particular attention should be paid to those muscles which have the most valuable individual function, namely, the hip extensors, quadriceps, deltoids and the intrinsic muscles of the hand. It should also be remembered that the abdominal muscles play an essential part in the maintenance of a good posture.

BIBLIOGRAPHY

- AUST, H (1935). *Physiother Rev*, 15, 183
 BRAHDY, M H, and LENARSKY, M (1934) *J. Amer med Ass*, 103, 229
 GALLOWAY, T C (1943) *J Amer med. Ass*, 124, 250
 GAUVAIN, H, and LANGSTON, H H (1939) *Int Bull. Econ. med Rev Publ Hyg*, A 40, 82
 KUBECEK, W G, et al. (1948) *Arch. phys Med*, 29, 84
 MORROW, D J, and STIMSON, B M. (1947) *Med Clin N Amer*, 31, 609.
 STEVENSON, F HARWOOD (1952) *Lancet*, 1, 845.
 STIMSON, B M (1940). *Laryngoscope (St Louis)*, 50, 57
 TOOMEY, J A. (1944) *Trans Coll Phys Philad*, 12, 14.

CHAPTER XI

DRUGS AND SERUM

I. DRUGS

PEIPER (1936) stated that against poliomyelitis and polio-encephalitis there is no known effective medicamental agent, and this is generally held to be equally true today, in spite of the eras of chemotherapy, penicillin and streptomycin. It is nevertheless my intention to describe briefly the drugs which have been, and are being used with varying degrees of success.

The pain in the acute phase may be effectively controlled by aspirin, phenacetin or codeine, whilst insomnia is treated by aspirin, potassium bromide or chloral hydrate in the appropriate dosage.

It is well known that the virus is very susceptible to oxidizing agents, and certain observers advocate the use of mouth washes and gargles with a weak solution of potassium permanganate.

Potassium chlorate in a 2% solution may be instilled in five minim doses into the nostrils three times a day. In addition five grains by mouth in twenty-four hours may be given to infants, and sixty to eighty grains to adults. This should be given immediately the diagnosis is made and should be continued for two to three days during the febrile period, being gradually diminished in dosage and discontinued at the end of six days.

Royle (1935) stated that a small amount of ephedrine, given in the preparalytic stage, prevented paralysis and oedema of the spinal cord in both experimental and human poliomyelitis, whilst Kubatsch (1937) considered that strychnine given intravenously is helpful. Ephedrine hydrochloride injected intraspinaly is said to reduce the local congestion and cause a local anaemia.

It should be pointed out that the above in our opinion is purely theoretical and is of no proved practical value.

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BIBLIOGRAPHY

- AUST, R (1935). *Physiother Rev*, 15, 183
 BRAHDY, M B, and LENARSKY, M (1934). *J Amer. med Ass*, 103, 229
 GALLOWAY, T C (1943) *J Amer med Ass*, 124, 250
 GAUVAIN, H., and LANGSTON, H H (1939) *Int Bull Econ med Rev Publ Hyg*, A 40, 82
 KUBECEK, W G, et al. (1948) *Arch phys Med*, 29, 84.
 MORROW, D J, and STIMSON, P M (1947) *Med Clin N Amer*, 31, 609
 STEVENSON, F HARWOOD (1952) *Lancet*, 1, 845
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 TOOMEY, J A. (1944) *Trans Coll Phys Philad*, 12, 14.

hours, and this should be increased to 1.5 units/kgm. eight hourly until all spasm has gone, unless there has been an adverse reaction. The object of this treatment is to establish and maintain normal muscle length by utilizing the relaxing effect of curare, and to enable the immediate institution of intensive physical therapy. By this means, patients are made ambulatory as soon as possible, thus preventing the loss of the kinaesthetic sense in the upright position. Exercises are usually started within twenty-four hours of admission to hospital, and no attempt is made to avoid fatigue. This method is, however, not without risk, as one of the twenty-nine patients died. The great disadvantage is that only a narrow margin of safety exists between an effective therapeutic dose and one which paralyzes the respiratory muscles.

Richards *et al* (1947) re-examined the claim of Ransohoff but found that they were unable to confirm that the administration of curare affected materially any aspect of the course of the disease nor did it shorten the convalescent stage. Jones and Dickson (1947) showed that the use of curare in sixteen cases had little effect on the paralysed muscles, but that the patients themselves appeared to be more comfortable. Rosenberg and Fischer (1948) found that the addition of curare for from three to ten days did not produce any more favourable results in the prevention of paralysis and disability than those which were obtained in previous years with the use of hot packs and other forms of physiotherapy alone.

The final word, however, has not yet been said about this drug, because recently it has been shown that if the motor nerves are crushed or divided in infected monkeys, the corresponding anterior horn cells will not be attacked, presumably because of the central effect of the peripheral disturbance. If these temporarily inactive neurones are immune from the disease, curare might be worth giving in the preparalytic stage, on the ground that a widespread damping down of activity at the myoneural junctions would reduce the danger of degeneration on the corresponding cord neurones.

Kabat and Knapp (1943) described the results of the treatment of twenty patients in the subacute stage of the disease with prostigmine, and concluded that the results were encouraging, and that the drug appeared to accelerate recovery. Apparently the range of passive movements was increased, the deformities decreased or eliminated by relaxation of the hypertonus, and in some cases active movement was increased. These findings were confirmed by Eveleth and Ryan (1944). Hodes (1947) examined the responses to the electromyograph in 14 patients and found them to be abnormal, but after the administration of prostigmine these responses became almost normal in 8 cases. On the other hand, Jones and Dickson (1947) treated 28 cases with prostigmine with little or no beneficial effect.

Rosenow (1939) reported on the failure of sulphapyridine to protect against experimental (virus) poliomyelitis. Rhett (1940), however, found that when 1 gr./1 lb. body weight of neoprontosil was given to 440 children with an acute infectious episode during an epidemic, only 3 cases developed paralysis or meningeal symptoms, and these cases had not kept up their maintenance dose. In 11 cases with paralytic poliomyelitis, the administration of the drug was followed by recovery from the paralysis.

Penicillin and streptomycin fail to give protection against

is fairly recent and has been published since 1947. Its use is based on the fact that the Sherrington Law of reciprocal innervation is altered, i.e. the antagonists of the affected muscles do not relax but remain in spasm, and this shortening leads to deformity. The paralysed group are consequently hindered in their recovery because of this opposition. It should be made quite clear that Sherrington's Law is upset by the disease and not by the administration of curare. Ransohoff (1947) advised the administration of 0.9 units/kgm. of body weight eight hourly for twenty-four

CHAPTER XII

NURSING

ONCE the physician has decided that hospital treatment is necessary, the patient should be transported in the recumbent position in an ambulance. A car should not be used, unless the patient can lie flat on a board which is adequately padded with blankets.

The nursing staff who are entrusted with the care of poliomyelitis cases must be highly trained, because apart from the general nursing care of the patient, they will require to take all necessary precautions to prevent the spread of the infection. In addition, they should be familiar with the deformities which may arise even in the earliest stages and with the splints and appliances which are employed to prevent or correct them.

Isolation of the patient, especially from children and young adolescents, is necessary for three weeks after the temperature has become normal. This is usually carried out in an isolation hospital by the pavilion or cubicle system, but unfortunately pavilions and cubicles are still relatively rare in Great Britain. Admission to an isolation hospital is preferable in the first place, because the patient may be suffering from some other infectious condition, and if no paralysis develops it is more economical in beds from the orthopaedic point of view. Another important point in favour of the isolation hospital is that the members of the staff are adequately trained in the intricacies of barrier nursing.

It is, however, essential that all proved cases should have adequate orthopaedic supervision from the onset of the disease.

It is quite usual for the patient to be nursed in a general hospital ward by barrier nursing, or preferably in a side ward, as quietness is an important part of the treatment. In the former case, a corner bed next to a window is to be preferred, as efficient ventilation helps to minimize risk of spread of the infection. The bed should be screened and the floor space

night and morning and the bedclothes are kept free from wrinkles and crumbs.

The limbs must be handled very gently and the joints supported when the patient is moved or when splints are removed.

In extensively paralysed patients, retention of urine with distension of the bladder should be watched for. In the early stages, retention may be relieved by raising the head of the bed on blocks, and by the application of heat to the abdomen. Next, if there is no response to antispasmodic drugs, e.g. carbachol, catheterization, tidal drainage or suprapubic drainage may be required. If there is incontinence, great care must be taken to prevent bedsores.

Constipation may be troublesome, but laxatives and cathartics should not be given. Repeated retention enemata of 2 to 4 ounces of warm olive oil and mineral oils, e.g. nujol by mouth are advised. Later when laxatives are permitted compound liquorice powder can be given. Incontinence of faeces usually occurs only in very severe cases.

Whilst using the bedpan, the body should be kept in a straight line, with the joints supported in the optimum position, but any splints in use should not be removed. The patient is turned on to his side and the bedpan placed in position. Folded blankets are placed above and below it, to support the trunk and lower limbs respectively. The patient is then turned gently on to the bedpan. Special beds or split mattresses assist greatly in its easy use.

To turn the patient on to his side, firm pillows or a folded blanket are placed alongside the body. The nurse supports the leg at the ankle and just above the knee, whilst an assistant supports the buttocks and shoulders. As the patient is turned gently, the leg and arm rest on the pillows, the arm being supported at the shoulder level, and the upper leg is brought slightly forward from the hip to avoid pressure on the underlying one. The pillow can later be made higher if there is much pain on moving the arm, thus preventing it from coming too far forward. If the abdominal muscles are weak and the abdominal wall sags, a firm supporting pad is indicated, as this invariably gives added comfort.

(It should be noted that these positions differ in certain respects from those advocated by Sister Kenny.)

This correct body and limb position must be maintained even in abortive and preparalytic cases. A careful watch must be kept to avoid pressure on the paralysed muscles by tight bandages and straps, or by the splints and appliances themselves.

The patient should be encouraged to take plenty of fluids, especially during the febrile stage, and the mouth is cleaned regularly. After the temperature becomes normal, a more varied nutritious diet, rich in vitamins, is given.

Some observers advise the use of ice-bags applied to the back of the neck and spine, and certain drugs to reduce the fever.

Pain may be relieved by the use of warm packs for five to fifteen minutes, or by the use of an infra-red lamp. Neuritic and neuralgic pains are usually relieved by sodium salicylate in doses of from ten to sixty grains. Sedatives may be given for restlessness, but only in very small doses because of the danger of respiratory paralysis during sleep.

A careful watch must be kept for signs of respiratory paralysis, the chief of which are restlessness, anxiety, insomnia, increased pulse rate, sweating, cyanosis, dilatation of the alae nasae and difficulty in speech.

Even in the mildest case of poliomyelitis, the patient should rest in bed for at least three weeks.

In the preparalytic stage, when there are signs of meningeal irritation, lumbar puncture is essential to exclude meningitis, encephalitis, etc., and preparations for this procedure must be made by the nurse.

In the preparalytic stage, when the diagnosis is certain, lumbar puncture may be harmful and is unjustifiable unless there is clinical evidence of increased intracranial pressure.

If there are no contra-indications, the bed may be raised at the end of three weeks, as this allows the patient to feed himself and to take an interest in what is going on around him. It also helps to increase his respiratory excursion. In these cases, raising the head of the bed has definite advantages over a back-rest.

To turn a child on to his face, he is moved *en bloc*, the pelvic and shoulder girdles moving together. The draw sheet which has previously been placed in the long axis of the body, is then drawn to the foot of the bed, so that the feet project through the bars at a right angle to the lower limbs. When in the prone position, one or two pillows should be placed under the chest and thighs so that the abdominal muscles are free to carry out the respiratory effort. Pillows should also be placed under the knees and feet to protect them from pressure.

If the child is restless, he is nursed on a straight Bradford frame or on a plaster bed. If on the former, he is turned on to his face with his feet over the edge of the frame which should be high enough to prevent the pressure of the bed on his toes. If on the latter, the patient can wear the anterior cast in the prone position, and the posterior cast in the supine position.

The nurse is responsible for ensuring that the position ordered by the physician is maintained so that stretching of the paralysed muscles is prevented. The position is maintained by sandbags, pillows or splints, and a bed-cradle is used to bear the weight of the bedclothes. Once again it must be stressed that rigid immobilization during the acute stage is never called for, as this invariably causes stiff joints and contractures.

The nurse must know how and why any apparatus is used, and a brief description of the positions to be maintained either voluntarily or by splinting is now given. A full description, however, is given in the appropriate section:

- (1) Head and neck kept straight.
- (2) Back straight.
- (3) Shoulder is abducted to approximately 60° .
- (4) Elbows are flexed to a right angle.
- (5) Wrists are slightly dorsiflexed with the fingers flexed
- (6) Thumbs are splinted in opposition.
- (7) Legs are kept straight with no medial or lateral rotation
- (8) Knees are slightly flexed.
- (9) Foot is kept at right angles to the lower limb.

respiratory or cardiac centres or from choking due to an inability to swallow. Aspiration of mucus and vomitus into the bronchi may cause aspiration pneumonia or a fatal bronchial obstruction, and it is therefore important to keep the pharynx free from secretions, food and vomitus

As excitement increases the flow of mucus, the patient

DIFFERENT FEATURES OF BULBAR PARALYSIS AND SPINAL-INTERCOSTAL PARALYSIS

Features	Bulbar	Spinal-intercostal
Pathology	Nerve cells of respiratory centre in medulla	Nerve cells of anterior horn of cervical portion of spinal cord
Respiratory muscles	No paralysis	Intercostals and/or diaphragmatic paralysed
Concomitant paralysis	Usually cerebral motor nerves and their muscles especially those of pharynx and palate	Usually muscles of shoulder girdle
Type of respiration	Irregular in rate and depth	Rapid and shallow
Speech	Nasal tone	Short sentences only
Dysphagia	Collection of mucus in pharynx	Absent
Mental state	Disoriented, often stuporous, but may be comatose	Apprehensive and anxious Rarely disoriented
Auxiliary muscles of respiration	Not improved and often made worse by the respirator	Immediate dramatic improvement

should be kept as quiet as possible Nausea associated with vomiting also causes increased secretions, but vomiting can be prevented by keeping the stomach empty. Fluids and carbohydrates are therefore given per rectum or intravenously in preference to nasal tube feeding

As severe attacks of coughing and choking weaken the patient, postural drainage should be tried, by raising the foot

A portable reading lamp is of great benefit, and in certain cases mirrors and prism spectacles can be used to permit even severely paralysed patients to read whilst remaining supine. Even patients with complete paralysis of both upper limbs can enjoy reading a book by the use of an electric page-turner.

The position of the bedside locker should be changed at intervals to prevent constant turning towards the same side, as this may contribute to torticollis or scoliosis if there is weakness of the neck, abdominal or back muscles.

Nursing Treatment of Cases with Respiratory Paralysis

The proper selection of patients for treatment in a mechanical respirator is essential not only from the point of view of the correct treatment, but also to economize in the use of respirators as during an epidemic the demand may sometimes exceed the available supply.

It is interesting to note that the first bulbar case treated by Drinker in 1929 died. Brahdý and Lenarsky in 1936 reported on 63 cases treated in the Drinker respirator, and pointed out that all these cases with involvement of the respiratory centre had died.

Nielson (1946) made observations on 110 cases with respiratory insufficiency of whom 76 were treated in the Sanlin (Swedish) respirator. The mortality was found to be greatest in those patients with signs of bulbar lesions. At post-mortem, there was emphysema of the upper part and atelectasis of the lower parts of the lungs, but only in the patients who were treated in the respirator.

It can therefore be concluded that the bulbar type of case should not be treated in a respirator if the respiratory muscles and the mechanics of respiration are still normal.

It has been suggested by Nielson (1946) that treatment in the respirator leads to the accumulation of carbon dioxide in the blood, and that the resultant acidosis contributes further to the fatal issue.

(1) *Respiratory Embarrassment Due to Bulbar Paralysis* — The patient is in danger of dying from involvement of the

cough, visible feebleness of chest and diaphragmatic movements and an inability to say one or two words with each breath (ask the patient to count). The patient is restless, irritable, fatigued and apprehensive. The accessory muscles of respiration, e.g. the sternomastoids and alae nasae are very active.

A mechanical respirator should always be "on hand" in the hospital, and it should be instantly available for any case with bilateral involvement of the shoulder muscles, cervical muscles and upper extremities.

In the case of young children, the respirator should, in appropriate cases, be used at the earliest possible moment, because if they survive the crisis, there is every possibility of recovery. Unfortunately this is by no means true for adults, for whom the prognosis is very much worse.

Patients may be treated in various types of respirator, e.g. the Both cabinet, the Sanlin, Drinker or a special orthopaedic type with a cabinet large enough to permit the use of splints for the support of the arms and legs, and so prevent contractures and deformities.

The importance of saving the life of the patient must not entirely obscure the necessity for protecting him from needless disability.

Certain observers prefer the Bragg-Paul pulsator in the late convalescent and residual stages, as the patient is then free to move about, and also it is readily transportable.

These respirators are, however, a mechanical means of preventing death from asphyxiation, and the type used is really of little importance. They may require to be used for the duration of the patient's life.

The Both Cabinet Respirator

As this is the usual type in use in this country, a brief description of the nursing technique will be given, as it is essential that the nurse should be entirely familiar with its mechanism.

Inspiration and expiration are both controlled in a box type of respirator, and the action is so powerful that it can overcome quite marked degrees of muscle spasm.

of the bed 30°. Drainage may also be aided by turning the patient on to his face, and by using a suction tube or an electrically driven suction apparatus. Because of the risk of trauma, the latter should not be used more often than is necessary to keep the patient relatively comfortable.

In extreme cases, tracheotomy may be necessary, and it is then the duty of the nurse to see that a free air-way is maintained.

If there is a failure of the respiratory centre, intravenous saline with 25% glucose every four hours for twenty-four hours and oxygen therapy have been recommended, and the nurse must have the facilities for these constantly available. Oxygen therapy by means of a nasal catheter or a B.L.B. mask can be administered in an emergency, but an oxygen tent is unsuitable. Lumbar or cisternal puncture may also be ordered to relieve the cerebral tension.

(2) *Respiratory Embarrassment Due to Paralysis of the Respiratory Muscles (Intercostals and Diaphragm)*—These spinal cases are ideal for treatment in the mechanical respirator but it can also be used in cases where the respiratory distress is due to the persistent spasm of the muscles of respiration, when the thorax will be found to be held in the position of expiration.

It is imperative that the late signs of respiratory embarrassment should not be waited for, before placing the patient in the respirator. The nurse should be warned to look for, record and report the slightest and therefore the earliest sign of impaired respiratory function, because they may last in the initial stage for only a very short time, and may be followed by a period of apparent normality.

The early use of the respirator helps to prevent the exhaustion of the weakened muscles, ventilates the lungs adequately, controls the acidosis and avoids a fatal result from anoxaemia.

There appears to be very little doubt that anoxaemia has a marked deleterious effect, and it may even induce a more rapid spread of the virus. The early signs of anoxaemia which must be looked for are anxiety, sweating, increasing restlessness, cyanosis and an increase in the pulse rate. There is also a quickening rate of respiration, loss of strength to

rubber neckpiece is then fitted comfortably and not too tightly over a bandage previously placed around the patient's neck to prevent chaffing by the collar. The neck may be protected with lanoline or petroleum jelly spread on lint and covered with gamgee tissue. The bed is then re-inserted into the cabinet, and the end cover is bolted into position.

Before starting the motor, the nurse should make sure that the air passages of the patient are free from mucus by aspiration if this is necessary. The motor is then started and a negative pressure of 12 to 18 ccm. of water is maintained by adjusting the valve on the top of the cabinet. This pressure gives an adequate ventilation of the lungs in a patient who has a complete paralysis of the respiratory muscles. In young children the negative pressure should be between 10 and 14 ccm. of water.

Air leakage around the collar can be prevented by using cotton wool packing.

If so desired, the cabinet can be tilted with the head downwards to approximately 20° from the horizontal, the patient's shoulders being protected with pads of cotton wool. If the diaphragm is weak, pressure of the mattress in the prone position increases the difficulty of breathing.

The temperature inside the cabinet should be kept just above the normal room temperature, i.e. 75° F.

As too much clothing obscures the movement of breathing, which must be under constant supervision, the minimum amount should be worn. An open-backed gown is advisable because of the ease with which it can be removed. Extra warmth can be supplied by bedsocks, well-protected hot water bottles, or by a lamp switched on inside the respirator.

Basic nursing care is essentially the same as for all patients, but a two-hourly schedule for changing the position of the patient should be organized. The number of assistants required for this depends on the position and the weight of the patient. Special care of the skin is necessary to prevent bedsores and excoriation around the neck by the rubber collar. The use of a rubber bedpan is most advantageous.

If the patient is of an age to understand, he should always be told that he is going to be nursed in the respirator, and

The machine is tested before use, and the speed of the artificial respirations regulated at 20, 26 or 32 pulsations per minute, according to the requirements of the patient. In our opinion, respiratory rates greater than 20 per minute are too high, and are likely to produce a condition of acapnia from the "washing out" of carbon dioxide. We therefore advise the maintenance of a slower rate of respiration than



FIG 9 —MAINTENANCE OF FREE AIR-WAY WHILST PATIENT IS IN BOTTL RESPIRATOR
(Reproduced by permission of the Nursing Mirror)

that which is usually recommended, and the administration of oxygen to overcome any anoxia. In an emergency, such as the failure of the electric current, or a breakdown of the electric motor, the bellows can be hand-operated at the rate of 20 strokes per minute.

The mattress and small pillow are prepared and a suitable rubber collar chosen. To insert the patient, the bed is pulled out, and the patient is placed on it, his head being near the oval opening in the end cover. His head is now put through this opening, and he is pulled in a horizontal direction until his shoulders come against the cover. The special sponge

therapeutic measure in order to rest the weakened muscles of respiration and to re-educate them towards normal power and balance. This may be achieved by putting the patient into the respirator for several hours a day, or he may be allowed to sleep in it. One of our patients who had extensive paralysis of the whole trunk and limbs, was nursed in an iron lung even during sleep. It was noted that respiration was carried on only by the accessory respiratory muscles. The vital capacity whilst lying was only 350 ccs. and when sitting 400 ccs.

Cases should also be treated in a respirator who show progressive weakening of the intercostal muscles, increasing respiratory embarrassment or fatigue of the overworked healthy muscles.

The respirator can also be used to treat insomnia which is due to dyspnoea.

The mechanical respirator must be discontinued gradually and this process may sometimes extend over several months. The tendency has been to remove the patient far too soon rather than too late. The effects of this early removal are cumulative over days or weeks, and the premonitory signs and symptoms are headache, irritability, insomnia, anorexia, and mild dyspnoea. These signs increase until an emergency arises which may result in the patient's death, or prolong his retention in the respirator for several weeks. The correct method is to open the porthole for varying periods and to gradually increase this time during sleep. During the intervals when the patient is breathing without the aid of the respirator, ■ careful watch must always be kept for the onset of sudden dyspnoea, which will necessitate the immediate restarting of the respirator, or the return of the patient to it.

Weekly estimations of the vital capacity of the patient should be carried out. This is an individual assessment, and other things being equal, the removal of the patient from the respirator can usually commence when the vital capacity has reached 750 to 1,000 cc.

It will therefore be seen that the mechanical respirator tides the patient over a critical period and may keep him alive until the respiratory muscles recover.

should be assured that it is only being used temporarily to give the respiratory muscles a rest. At the beginning, the patient may be apprehensive until his breathing becomes synchronous with that of the respirator, and when this is established, he usually falls into a peaceful and restful sleep. Synchronism may be established by increasing temporarily the negative pressure or by increasing the rate of the pulsations. If breathing is not synchronous with the respirator after five minutes, involvement of the respiratory centre must be suspected. If there is spasm of the chest muscles and diaphragm, hot packs may be applied to the thorax through the portholes. It is important to remember that the pressure should be increased to 25 to 30 ccm. of water for five minutes several times a day, so that the lungs may be fully ventilated.

During the acute stage, it should be possible to carry out all nursing treatment without stopping the machine. The opening of a porthole should immediately follow an inspiration, but the porthole should be closed as soon as possible. Certain observers advise the administration of pure oxygen by mask for five minutes before the portholes are opened and that it should be continued until they are closed. If the cabinet is open for any length of time, such as for changing the bedclothes, accessory artificial respiration can be given by the McKesson resuscitator or by a Boyle's anaesthetic apparatus. In the latter case, the lungs are inflated by the rhythmic compression of the rubber bag whilst the face mask is closely and firmly applied.

Eve's tilting stretcher or manual methods may sometimes be used in relatively mild cases, whilst the patient is having orthopaedic treatment of short duration, out of his respirator, but they should not be continued for long periods as they are very distressing to the patient.

The respirator may be used continuously for days, weeks or months, thereby protecting the respiratory muscles from undue or cumulative fatigue. In this way, recovery may be more rapid and complete especially in cases where there is only a mild or moderate weakness of the respiratory muscles.

The mechanical respirator may also be employed as a

therapeutic measure in order to rest the weakened muscles of respiration and to re-educate them towards normal power and balance. This may be achieved by putting the patient into the respirator for several hours a day, or he may be allowed to sleep in it. One of our patients who had extensive paralysis of the whole trunk and limbs, was nursed in an iron lung even during sleep. It was noted that respiration was carried on only by the accessory respiratory muscles. The vital capacity whilst lying was only 350 ccs and when sitting 400 ccs.

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It will therefore be seen that the mechanical respirator rides the patient over a critical period and may keep him alive until the respiratory muscles recover.

VITAL
CAPACITY
3200

2800

2400

2000

1600

1200

800

600

400

1

2

3

4

5

6

7

8

9

10

11

12

MONTHS

FIG 10

passive movements

minimal activity

exercises in pool

standing in pool

walking on dry land

discharged to O.P.D.

back to
work

In rare cases, with abductor laryngeal paralysis, tracheotomy and/or oxygen therapy may be required in patients who are being treated in a respirator.

One last point which is sometimes forgotten is that the respirator must be adequately serviced, and there must always be an adequate and immediate supply of spare parts

The Stanco Respirator

In this country the vast majority of hospitals dealing with cases of poliomyelitis are equipped with cabinet respirators of the Both type. In recent years Stanley Cox Ltd have perfected the very latest type of respirator—the Stanco, which incorporates many new features. The whole apparatus, in cream enamel and chromium plate, has been carefully finished to eliminate as far as possible the “coffin” appearance, which undoubtedly had an adverse psychological effect upon most patients. Every consideration has been given for the comfort of the patient, including a very comfortable Dunlopillo mattress, four strip lights which provide both light and adequate heat and a large port provided with

quick action fasteners for the insertion of bedpan or urine bottle. There are also five miniature ports for the introduction of feeding tubes, four arm ports on each side of the cabinet and two large perspex windows on the top of the

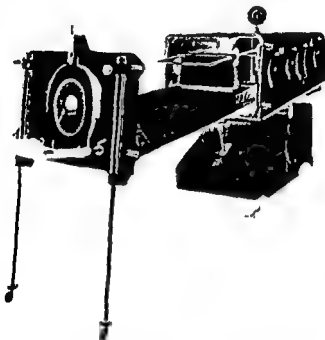


FIG. 11 —STANCO RESPIRATOR OPEN

cabinet which allow full inspection of the patient. The pulsator unit can produce a negative pressure as high as 35 cms. of water, whilst positive pressure up to 10 cms. of water is controlled by a separate valve. A simply operated device warns the attendant of pressure failure due to any cause

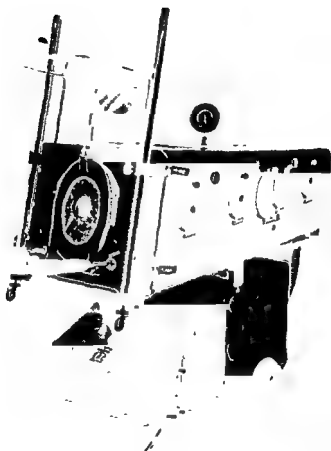


FIG. 12 —STANCO RESPIRATOR CLOSED

Cuirass Respirator

This is not so certainly effective that it can be depended upon for the treatment of the initial stage of acute respiratory muscle paralysis when paralysis may quickly progress, but may be used as an adjunct to the tank machine. It is useful during the process of weaning a patient from the respirator, for the chronic care of moderately paralysed patients, and when a tracheotomy is necessary.

The Bragg-Paul Pulsator

This apparatus can be used even in young children, as different sizes of air-belts are supplied. Air is forced into the chest belt at definite rhythmical pressures by electrically

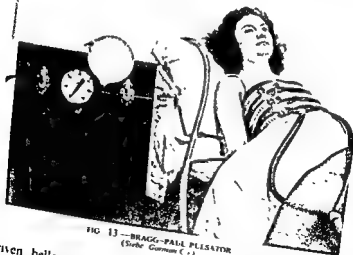


FIG. 13.—BRAGG-PAUL PULSATOR
(Siebe Gorman Co.)

driven bellows. Each pulsation produces an involuntary expiration, and the release of the pressure allows the natural elasticity and recoil of the chest and diaphragm, with a resulting inspiration.

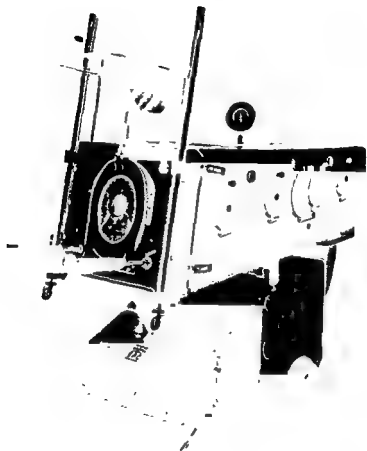


FIG. 12—STANCO RESPIRATOR CLOSED

Prolonged recumbency in a patient may result in renal calculi which may cause death from renal insufficiency. This is treated prophylactically by

- (1) Regular and frequent changing of the position of the patient
- (2) The administration of abundant fluids
- (3) The reduction of calcium and vitamin D in the diet
- (4) The prevention and control of urinary infection.

As the patient often suffers from a prolonged illness, he may become irritable and apprehensive. The nurse must therefore show patience, understanding, sympathy, tact and great gentleness, as this helps to dispel fear and establish confidence

On discharge of the patient, the nurse should make sure that both he and his relatives understand fully the fundamentals of nursing care required in his particular case and that instruction has been given in the application and the care of appliances, etc

BIBLIOGRAPHY

- BRAIDY, M B, and LENARSKY, M (1936) *J Pediat*, 8, 420
BRUCE, J W (1941) *Kentucky med J*, 39, 518.
NIELSON, E M (1946) *Ugeskr Læg*, 108, 1341.

One disadvantage of the Bragg-Paul pulsator is that it assists only during the passive phase. Movement of the tender and painful intercostal muscles by the pulsator may cause them to pass into spasm. Reflexly this spasm causes a marked variation in the respiratory rate, but the apparatus cannot control the constantly changing respiratory rhythm.

The pulsator works at a rate of approximately 14 to 16 pulsations per minute and it can also be worked by hand. It is useful in cases with a very low vital capacity and the patient can sleep in it. Positive pressure oxygen therapy may be required if there is any suspicion of pulmonary oedema.

Blockage of the bronchi and collapse of the lung may result from bronchial catarrh. This is best treated by penicillin, postural drainage, gentle manipulations and frequent change of position. Atropine sulphate and sedatives are definitely contra-indicated.

CONVALESCENT STAGE

During the convalescent stage, the patient must receive an adequate amount of fresh air and sleep, and mental and physical fatigue must be avoided. The skin and muscles are kept warm and the correct posture maintained by the use of any necessary appliances.

The patient is given general tonics and an easily-digested nourishing diet.

The early symptoms of anoxaemia in a patient who has been in a mechanical respirator for several weeks or months are insomnia and headache, and they should be treated by the administration of oxygen.

Bruce (1941) drew attention to the fact that vitamin B is supposed to increase the resistance of the nerve cells, but its true value in this respect is still not definitely proved. Vitamin B does, however, improve the carbohydrate metabolism in the nerve cells and will therefore produce a more satisfactory oxygenation of pyruvic acid. Vitamin E is also given as it is said to improve the muscle tone, decrease capillary permeability and counteract the development of fibrosis.

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BIBLIOGRAPHY

- BRANDY, M B, and LENARSKY, M (1936) *J Pediat*, 8, 420.
BRUCE, J W (1941) *Kentucky med J*, 39, 518.
NIELSON, E M (1946) *Ugeskr Laeg*, 108, 1341.

CHAPTER XIII

MUSCLE RE-EDUCATION

I PROPOSE to give only a brief description of muscle re-education because this form of treatment should preferably be given by a physiotherapist who has been specially trained in the treatment of poliomyelitis.

It must first of all be pointed out that it is a mistake to imagine that the patient must keep the limbs active to counteract a spreading of the paresis. The patient should be left entirely alone until the maximum paralysis has developed, and this usually takes three days. The only treatment necessary during this waiting period is to apply the appropriate splints to prevent any tendencies to deformity.

In performing a voluntary movement, all the intact muscle fibres do not necessarily contract, because the muscle can adapt itself to the amount of work it has to perform, and the more vigorous the exercise, the greater will be the number of fibres involved. When the function of a motor nerve is completely or partly destroyed, attempts are still made to produce the movement by sending impulses to the region along other intact nerves.

The main aim of muscle re-education is to produce hypertrophy of the remaining muscle fibres in an attempt to compensate for those which have lost their nerve supply. The treatment should also be directed towards maintaining good alignment both in weight bearing and in non-weight bearing. Controlled muscle activity and exercises to the unaffected part of the body are also given to maintain muscle tone and to prevent disuse atrophy, and they should be given as soon as muscle tenderness disappears.

There is no doubt that physiotherapy treatment should begin as soon as possible, because delay may be partly responsible for the development of contractures and an unnecessarily long prolongation of the convalescent period.

It is essential that right from the beginning and throughout treatment that the physiotherapist should have the full

co-operation and confidence of the patient and keep an accurate record on the patient's muscle chart

Whatever method of treatment is used, recovery will be extremely rapid if there is only a transient weakness of the muscle group

Forrester-Brown (1938) stated that muscle re-education should begin immediately a "flicker" movement appears, and that the first consideration is passive and guided exercises.

FUNDAMENTAL PRINCIPLES OF KINESIOLOGY

A muscle contracts in overcoming resistance, and with increasing tension it shortens—concentric muscle work. A muscle contracting can also be called upon to resist whilst its state of tension gradually diminishes as the muscle lengthens—eccentric muscle work. Both concentric and eccentric muscle work are essential in the early stages for aiding returning muscle power. Manual control and resistance can easily be graduated to correspond with the strength of the weak muscles

Types of Muscle Action

(1) *Prime Movers* These are muscles concerned with performing a movement, e.g. the deltoid. These should take first place in a scheme of muscle training.

(2) *Synergists*. These are muscles which prevent undesired action. An understanding of synergist action is most important in the study of paralysis because of the effect that a muscle can have upon the action of another.

(3) *Fixators*. These are muscles which steady a part. They are often required to use the greatest amount of energy and adequate fixation must be obtained before maximum strength can be exerted.

(4) *Antagonists*. These are muscles which reciprocally relax co-operating fully with the prime mover as it contracts. It can be used to obtain muscle relaxation and to avoid tick movements and when it is necessary to stretch tight muscles.

(5) *Tonic Contraction*. This is a state of mild muscle contraction concerned with the maintenance of posture.

Stretch Reflex

In stretching a normal muscle, there is an increase of tension in that muscle. When the stretch ceases, the muscle relaxes. This is a reflex response and can only be obtained in the gravity resisting muscles. The stretch reflex varies with the state of tone in a muscle and is useful in evoking active contractions in early muscle training.

To re-educate the impaired impulses of movement, a muscle or muscle group must be put through its full arc between full contraction in flexion and full extension, first passively to establish a mental and visual picture of the movement to be taught. Once the co-operation of the patient has been gained, active contraction of the affected muscle should follow, the patient concentrating on that movement which is being executed under the guidance of the physiotherapist. In the later stages, the muscle activity should be gradually increased, by decreasing the assistance given and increasing the number of movements and the resistance. The whole purpose of these exercises is to strengthen the weakest muscles.

Active contraction of the muscle is first obtained in the middle range of the movement, and if the inner range cannot be performed, it should be completed as an active assisted exercise. In the early stages of training, the middle and inner ranges should primarily be used. Outer range movements should be done only when the patient can carry out those in the inner and middle ranges. The position for the exercises varies according to the strength of the muscle and the function which can be elicited from it in that particular position. Maximum strength and the rate of fatigue under different conditions should be recorded. The weakened muscles are required to function without allowing neighbouring strong muscles to substitute for their action, and care must be taken that the paralysed muscles are never over-stretched. The tendency to adopt certain deforming postures must be avoided.

The most useful muscle activities are the mass movements rather than the action of a particular muscle or muscle group.

Patients have been known to make very good progress when their muscles are well co-ordinated even although accurate testing has shown very little muscle power.

Leverage

The principle upon which leverage is based in the human body is to maintain balance and to aid the movement required from all the various situations met with throughout life. There are three orders of levers. The first order is represented as the movement of the skull on the atlas. The skull is the weight and the atlas the fulcrum, and the muscles of the neck represent the power by which the head is moved on the vertebral column. The second order is represented as the foot which moves as a lever in walking. The third order of levers is employed particularly to increase speed of movement. A weight in the hand is the load to be moved, the elbow joint the fulcrum and the biceps and brachialis muscles represent power.

The majority of muscles in the human body appear to work as the third order of leverage.

In poliomyelitis, the physiotherapist makes use of the short or long levers according to the strength of the muscles involved in the movement. In training a weak deltoid, with the elbow joint flexed, the weight arm is shortened and movement is easier. As the muscle strengthens, the elbow joint may be fully extended thus increasing the mechanical disadvantage to the deltoid. In testing weak muscles, the most favourable circumstances as regards leverage are when the muscle acts with the least obliquity.

i. In the early stages, muscle re-education can be used in the following progressive methods:

- (1) With gravity assisting the movement. Here the movement is performed in a "downhill" direction.
- (2) With gravity eliminated from the movement.
- (3) With gravity resisting the movement. The movement takes place in a vertical "uphill" direction.

The full effect of gravity is involved in movements of muscles acting on the wrist, elbows, shoulders, neck, ankle,

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In stretching a normal muscle, there is an increase of tension in that muscle. When the stretch ceases, the muscle relaxes. This is a reflex response and can only be obtained in the gravity resisting muscles. The stretch reflex varies with the state of tone in a muscle and is useful in evoking active contractions in early muscle training.

To re-educate the impaired impulses of movement, a muscle or muscle group must be put through its full arc between full contraction in flexion and full extension, first passively to establish a mental and visual picture of the movement to be taught. Once the co-operation of the patient has been gained, active contraction of the affected muscle should follow, the patient concentrating on that movement which is being executed under the guidance of the physiotherapist. In the later stages, the muscle activity should be gradually increased, by decreasing the assistance given and increasing the number of movements and the resistance. The whole purpose of these exercises is to strengthen the weakest muscles.

Active contraction of the muscle is first obtained in the middle range of the movement, and if the inner range cannot be performed, it should be completed as an active assisted exercise. In the early stages of training, the middle and inner ranges should primarily be used. Outer range movements should be done only when the patient can carry out those in the inner and middle ranges. The position for the exercises varies according to the strength of the muscle and the function which can be elicited from it in that particular position. Maximum strength and the rate of fatigue under different conditions should be recorded. The weakened muscles are required to function without allowing neighbouring strong muscles to substitute for their action, and care must be taken that the paralysed muscles are never over-stretched. The tendency to adopt certain deforming postures must be avoided.

The most useful muscle activities are the mass movements rather than the action of a particular muscle or muscle group.

injury to the epiphysis, and gentle force should be used. Adequate relaxation of the shortened muscles can be obtained by the use of reciprocal inhibition. The patient must be taught actively to contract the opponent muscles so that the muscle to be stretched will be reciprocally relaxed, otherwise the tight muscle will be thrown into spasm. It is necessary to overcorrect at each manipulation.

Splints and apparatus can sometimes be used when it is necessary to maintain gentle stretching of a continuous nature.

TECHNIQUE OF TREATMENT

No definite rule can be followed in connection with muscle training of a poliomyelitis patient.

The aim of treatment is to re-educate voluntary contraction in the affected muscle by individual and co-ordinated action, and to encourage activity by increasing the frequency of an exercise or making the muscle task harder.

The decision as to whether to increase the task of the muscle depends upon its ability to move the joint smoothly through its full range. It is therefore wiser to increase the frequency of the exercise until full range has been achieved. The last movement should be as well performed as the first.

It is essential to have the part under training stripped for observation. Fatigue must be avoided and it is more noticeable in a lessening of the muscle effort. Warming the tissues prior to performance is indispensable and a warm pool or radiant heat are both effective.

It must be assumed that no muscle is completely denervated, and therefore muscles which are apparently paralysed must be given training equally with recovering muscles.

A muscle which has had adequate treatment for a period of six months and which still remains paralysed will not subsequently regain any recovery of functional significance with the passage of time. All muscles except those of no apparent power should receive whatever progressive training is necessary to achieve maximum strength, and it may be necessary to continue for as long as eighteen months or two years after the onset.

knee and hip Tests for the back and abdominal muscles must also be assessed according to the gravity factor.

Gravity can greatly aggravate an already disturbed balance, caused by asymmetrical weakness. Some types of deformity can appear with amazing rapidity.

Muscle Stretching

During the treatment of poliomyelitis, it may be necessary to stretch a muscle for the following reasons:

- (a) To initiate reflex tension in a muscle during the early stages of muscle re-education. Stretch reflex in a normal muscle responds reflexly to stretch by an increase in tone. This condition antagonizes the stretching forces in an effort to maintain equilibrium. The greater the stretch, the greater the contraction. Continuous stretching will cause damage and a muscle whose nerve supply is wholly or partially damaged can be greatly harmed by continuous stretching. No harm, however, appears to result from the use of a gentle stretching of a weak muscle during training. The increase in tension acts as a stimulus to recovery.
- (b) To stretch contractures and shortened muscles. A muscle becomes shortened when its origin and insertion are brought nearer together. It may arise as a protective mechanism through pain and tenderness being present in the limb. If the position is fixed for a prolonged period, real shortening will result and the ligaments and joint capsules are thought to become affected by fibrosis resulting in severe contractures. Similarly when muscles which surround a joint are paralysed, the ligaments become stretched and the joint subluxated. This is particularly noticeable in the shoulder joint.

Stretching Manipulations

Before commencing treatment, the patient must be placed in a comfortable position and the part should be warmed at least twice daily, if necessary three or four times a day.

The limb is supported close to the joint so as to avoid

injury to the epiphysis, and gentle force should be used. Adequate relaxation of the shortened muscles can be obtained by the use of reciprocal inhibition. The patient must be taught actively to contract the opponent muscles so that the muscle to be stretched will be reciprocally relaxed, otherwise the tight muscle will be thrown into spasm. It is necessary to overcorrect at each manipulation.

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Training of a Muscle of No Apparent Power

The part to be exercised must be supported, carrying out the movement passively whilst the patient concentrates on *the attempted movement*, thus the impulses of movement are awakened, and the assistance given by the physiotherapist must synchronize with the patient's mental and physical effort.

To initiate reflex tension in the muscle, gentle stretching should be given from time to time. Passive movements through the full range should be repeated several times during treatment, and in the majority of cases a contraction is seen after two to three weeks. The patient must be taught to watch the contraction, learning the feel of tension, and his full attention must be concentrated on the anticipated movement. The physiotherapist must assist the movement, aiming to obtain the patient's maximum concentration in the middle range by lessening assistance at this stage. As muscle power returns, co-ordinated exercises must be introduced, but *resistance must not be given until the muscle is able to perform the movement smoothly through its full arc*. This should be taken as the criterion for further progressive resistance.

The judicious use of varied technique helps to avoid boredom for the patient during many weeks of treatment.

(1) Manual control in the form of assistance or resistance is conveniently used in the early stages of muscle re-education. The control can be graduated to correspond with the returning muscle power, each movement being undertaken with adequate support of the part to avoid joint strain. The change in leverage which takes place during movement is a guide to the amount of resistance to be given. The middle range is easier to achieve than the inner or outer range, therefore the resistance should be lightly applied at the beginning and the end of the arc of movement, and more strongly applied in the middle range. The resistance given should be slightly less than that which would stop movement.

The disadvantage of manual control is that both hands are occupied in supporting the limb so that the operator cannot be certain that the required muscles are contracting.

(2) The re-education board. This technique requires a large plywood board about 3 feet 6 inches square with an even-polished surface. This board is placed under the part to be exercised so that the limb rests on it, support is adequate and friction is greatly reduced by the polished surface. The physiotherapist guides the limb with one hand, steadying the fulcrum with the other hand.

For the upper limb the board can be used for the re-education of the deltoid muscle. The board is placed in the nature of a backrest, the shoulder is fixed with one hand whilst the other guides the arm in abduction. A short or long leverage can be made use of by either fixing the elbow or the arm can be more fully extended. The re-education board can be placed on an inclined plane where gravity may assist the movement which takes place in a "downhill" direction or gravity may resist the arc of movement which takes place in an "uphill" direction.

For re-education of muscles in the lower limb in adults, trick movements are difficult to prevent.

(3) Suspension therapy. The apparatus required is simple, cheap and can be speedily erected in a limited space. Care is required in the selection of suspension apparatus. A strong supporting frame should be of adequate height with slings and ropes of suitable strength.

The advantage of suspension therapy is that it leaves the operator's hands free, one hand being used to control the range of movement whilst the other is free to palpate the tendon of the acting muscle. Assistance or resistance to the movement can be given manually by the operator. Friction is eliminated and gravity is counter-balanced except as is explained later by changing the fixation point of suspension. Gravity can assist or resist muscle action. Trick action can be more easily detected and prevented. Isolated muscle groups can be re-educated whilst neighbouring muscles remain relaxed. The psychological effect on a comfortably suspended patient is marked; once he is aware of the rhythm of motion he can pick up and follow movement more easily, thus encouraging greater effort. To obtain an accurate analysis of the power of trunk muscles, partial suspension can be

used with advantage. One half of the body is lowered to rest on a fixed point whilst the other half is raised in suspension. Partial suspension can also be used for strengthening the weak trunk muscles.

The Principles of Suspension Therapy

There are three different types of suspension.

(1) *Axial Suspension.* The limb is supported in slings, the ropes of which are fixed at a point above the joint which is to be moved. The limb can therefore move in one plane from one axis in perfect balance. Axial suspension allows the fullest range of joint movement.

(2) *Pendular Suspension.* For this type, the axis of movement is moved to a point away from the joint. Friction is therefore not vertical, but the plane is truly horizontal.

The advantage of this type of suspension is that a particular muscle group can be activated whilst its antagonist muscles remain relaxed. By altering the fixation point, it is possible to obtain either assistance or resistance by gravity for the acting muscles, and the suspended limb will swing back to the fixed point. Only a half of the arc of the swing is used to give either uphill or downhill effect, and the limb then swings back in relaxation to the fixed point. Pendular suspension can be of great assistance in the early stage of muscle re-education. The patient can watch the movement and can be taught to synchronize thought and effort whilst the operator initiates a gentle pendular swing.

(3) *Spring Suspension.* This allows movement in multiple axes and planes and may be used to assist movement by making use of the recoil action, and timing the recoil exactly with muscle effort.

The patient is suspended in the prone or supine position and can be taught co-ordinated exercises such as swimming and natural movements which are similar in character.

Co-ordination may be taught by rhythmical exercises to music. This is especially important in children who can associate certain movements with the music they hear.

In partial paralysis of the diaphragm and intercostal muscles, breathing exercises should be given daily and the

vital capacity recorded every two weeks. Poor breathing habits cause bad posture, sleeplessness and dullness, and patients should therefore be taught to breathe deeply and rhythmically by the physiotherapist.

It must also be noted that alterations in protective supports are needed fairly regularly in children and adults to meet any changes in the muscle balance, but appliances should never be discarded without the permission of the orthopaedic surgeon.

Physical treatment may continue for two to three years, and during this period it must be constantly supervised and adapted as the muscle power is regained.

Weak muscles begin to increase in strength within two weeks of the onset of the paralytic stage. Recovery is most striking during the first twelve weeks, but gradually slows down, and after approximately two years of supervised treatment, it is doubtful if further recovery is possible. It should, however, be pointed out that a patient who has not had supervised treatment from the beginning of his illness, may show an increased rate and degree of improvement subsequent to the substitution of these exercises, and cases have been recorded where this has occurred after a period of as long as ten years. On the other hand, a limb which has recovered imperfectly from an old poliomyelitis paralysis may show an increased weakness after several years. This is probably due to the excessive use of the weakened muscle, and it is more commonly noticed in the arm than in the leg.

Permanently and completely paralysed muscles should be recognized as soon as possible. If there is no trace of contraction after three months, it will never recover any useful function.

Thermotherapy

The metabolic rate of the tissues is increased locally and directly by heat. The effect of the increased temperature of the limb is to warm both the nerves and the muscles. As a result of this:

- (a) The threshold of the nerve, the neuro-muscular reaction

used with advantage. One half of the body is lowered to rest on a fixed point whilst the other half is raised in suspension. Partial suspension can also be used for strengthening the weak trunk muscles.

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cedes other physiotherapy treatment. The mild heating aids in softening contracted tissues so that the range of movement can be improved.

power of the muscle. Certain observers nevertheless consider that the application of heat in any form does not relieve the muscle spasm. The application of two-hourly packs and re-educative movements as advocated in the Kenny treatment appears to us to be too exhausting for an acutely ill patient.

The cold cyanosed limbs which are a frequent result of severe paralysis can be helped because the moist local temperature produced by the above treatment increases the blood flow to the limb.

It has already been stated that the efficiency of muscle action is improved by heat and therefore warming the tissues before exercise is indispensable to the performance of maximum muscle effort.

Pool Therapy

This is a form of treatment devised to avoid too early specific muscle re-education, by the partial elimination of gravity by the water. It can also be used for general function and balance exercises, swimming exercises, training in walking and in diversional games.

Pool therapy allows great diversity of movement of the joints, and it is usually carried out in a Hubbard's tank or a treatment pool. The Hubbard's tank is ideally suited for individual cases and for specific muscle training, but in units dealing with a large number of cases, a special pool is necessary. This pool should be of such a height above ground level that the physiotherapist can comfortably and conveniently control any particular movement. She must always be able to reach the patient throughout the treatment and to support the limbs when necessary. In the pool, there should be two shallow bays, one 12 inches deep for children

to stimuli is lowered and also the muscle threshold itself is lowered.

- (b) The duration of the muscle contraction is shortened.
- (c) The refractory period of the muscle is shortened.

In other words, the irritability of the muscle is increased. Although the exact mechanism is still not definitely known, there is no doubt that thermotherapy has the following beneficial effects:

- (1) *It relieves pain and discomfort which are often considerable in the early stages.*
- (2) *It improves the circulation.*
- (3) *It stimulates metabolism.*
- (4) *It improves the efficiency of the muscles.*

Muscle tenderness can be relieved by moist heat in the form of hot packs. One observer advocates the use of hot packs to relieve the muscular discomfort and pain, and they should be applied to all tender muscle groups for half an hour twice a day. They should be discontinued when the muscle sensitivity disappears. Prolonged acute muscle tenderness lasting for six to eight weeks appears to respond more readily to frequent hot packs applied two-hourly.

We have found that a Hubbard tank in which the water is kept at 100° F is most useful in the sensitive stage. The patients are able to relax and so obtain great comfort.

Other observers recommend the use of dry heat in the form of infra-red irradiation. Infra-red rays relieve muscle tenderness if applied in the early stages. The effect of these rays is due to the fact that they penetrate the skin producing profound hyperaemia locally, dilating the blood and lymphatic capillaries, and having a sedative effect on the nerve endings.

Radiant heat administered by a radiant heat lamp or by a cage large enough to cover the affected area can also be used.

Our experience has shown that carefully administered local heat in the form of short wave diathermy is particularly valuable for muscle tenderness and stiff joints, when it pre-

assisted exercises can be given to all the paralysed muscle groups. As a guide to early treatment, observers suggest that each active assisted movement should be directed three times in the beginning, increasing the number of each specific movement according to the condition of the patient, the physiotherapist watching carefully for signs of fatigue.

Patients with flail limbs can be taught balance and walking exercises in a treatment pool, by firmly supporting the knee joint with light metal or perspex gutter splints bandaged to the limbs. For balance and walking exercises it is necessary for the water level to reach the costal margin, if the patient is to receive adequate support from the water. Buoyancy can be used for the assisted movements in the early muscle training, and later it can be used as a resistance in the progressively stronger exercises. A simple toe-raising sling attached by an elastic strap below the knee joint is adequate for the purpose of supporting a dropped foot.

Lowman (1933) showed that this form of treatment offered complex possibilities for gradually increasing muscle activation, and that it was a good guarantee of the care of the muscles. In 1935, Lowman stressed the great importance of early pool therapy in the treatment of trunk muscles, and pointed out

that it can be done. He attributes this to the increased activity of the stronger muscles, whereas the actual treatment should be concentrated on these weaker muscles. He also points out that if the physiotherapist has not carefully analysed the muscle involvement of each patient, then swimming strokes usually given to strengthen muscles already strong, e.g. pectoralis major, will increase their tendency to contraction or the overaction against weakened opponents, e.g. the deltoid, and so may increase the deformity. We have not, however, met this complication in our unit.

A careful watch must be kept so as not to overwork the patient, and any diminution of power due to fatigue of the affected muscles must be appreciated and treated appropriately. It is therefore wise to limit the period of time in the

and the other 18 inches deep for adults. Part of the pool should be of a suitable depth, width and length to allow a free area for walking and swimming exercises and for games. The ideal pool should have two walking lanes each of which should be fitted with a set of parallel bars at a suitable height for children, possibly on a built-up base, and another set at a convenient height for adults.

In some treatment pools, in America and in this country, the physiotherapy staff go into the pool with the patient. This is essential if the pool is deep or very large, otherwise the physiotherapist would not have full control of the patient.

Warm fresh water or hypochlorite solution at a temperature of 90° F. is used for the less severe cases, but the temperature may be raised to 100° F. for spastic cases.

This form of treatment should be started as soon as possible and usually begins within seven to twenty-one days from the onset of the illness. The warmth and buoyancy of the water is comforting to the patient with muscle tenderness and stiff joints and encourages him to make an effort for himself. Thus the mental relaxation banishes apprehension and inhibitory reactions such as spasm. In short, the whole nervous system is relaxed, the peripheral vessels are dilated, and there is a marked sedative effect on the peripheral sensory nerves.

The patient must be transferred carefully to the Hubbard's tank or treatment pool. He should be placed in the water slowly and should be allowed to rest for a short period before treatment is commenced. This allows him to adjust himself, and is particularly important if he appears anxious or if he has respiratory weakness. He should be supported comfortably so that relaxation can be obtained, and he may require to retain the splints throughout treatment in cases which have been operated upon, or when the limb is extensively paralysed. In other cases, the paralysed parts may be supported by rubber rings or corks, but in the majority of cases it is quite safe to remove all splints and supports during immersion. As friction and gravity are now virtually eliminated, the exercises are mainly effortless, and active

which may retard its recovery or make complete recovery impossible.

Passive stretchings should be retained throughout early treatment in order to correct any tendency to deformity, and it is advisable to keep a record of the range of passive movements at least until after the full range of joint movement has been obtained

Active Movements

(1) ASSISTED MOVEMENTS

Assisted movements are necessary when a voluntary contraction of any particular muscle or muscle group is charted as 0, 1, or 2, that is to say, when the active effort is unable to overcome gravity.

Assisted movements can be obtained in a treatment pool where the buoyancy of the water provides helpful assistance in early movement. Sling suspension in which the limb is supported in slings also eliminates gravity. In some hospitals, a flat powdered board is used to obtain gravity-assisted exercises, the limb to be exercised resting comfortably upon the flat surface.

In the early stages, manual assistance is the best method, as the physiotherapist can immediately and accurately control the amount of assistance given to correspond with the effort of the patient.

The patient is instructed carefully in the movement required, either on his sound limb or by a personal demonstration by the physiotherapist. The movement is then carried out on the paralysed limb, being assisted by gravity or by the physiotherapist. The limb is gently and slowly carried through the motion which represents its function, whilst the patient at the same time makes every effort to use the muscles.

In the early convalescent stage, the primary action of the individual muscles or single muscle groups should be the first to be re-educated.

Assisted movements should be continued until the patient can voluntarily and without assistance hold the limb in the desired position by full contraction of the affected muscles.

If contractures and stiff joints are already present when the patient first comes under skilled supervision, the shortened tissues should be gently but thoroughly stretched at frequent intervals. Warm baths or irradiation with luminous heat can be of great assistance in softening the contracted tissues before stretching is begun. All movements should be very gentle and the limb must be completely supported throughout the exercises. The limb should be grasped firmly but gently over the joints. The arm is supported at the wrist and elbow, and the leg at the knee and ankle. In this way, pressure on tender muscles is avoided. Gentle passive exercises should be given twice daily. Particular care is taken with joints which have asymmetrical muscle weakness. It is *most important to note that the movement must oppose the deformity positions*. Constant supervision must be given to patients with a tendency to develop a particular deformity. Certain observers have stated that if an abnormal range of movement is permitted, or if an extremely paralysed limb is not supported, stretching or weakening of the joint capsules and ligaments will result.

Vigorous irresponsible stretching may result in muscular tears. This leads to the formation of scar tissue and adhesions which further limit movement.

Severely weakened bodily segments may result in instability of these areas and permit deformities to occur which may be far more disabling than the contractures.

Our experience has shown that movements of the interphalangeal and metacarpophalangeal joints of the hands and feet and the full range of movement of the spine tend to be the most difficult to correct once these joints have become stiff. Full extension of the elbow and dorsiflexion of the wrist and ankle are also most important.

Certain observers advise careful passive stretching of the weakened and paralysed muscles after the painful stage of the disease has passed in order to establish the proprioceptive sense. This treatment should be continued until the muscle tends to remain supple and of full length.

It is hardly necessary to mention that it is definitely harmful to retain a muscle in an overstretched position,

attempt should be made to get the muscle to work at its maximum strength

Resisted exercises should be of a specific nature, because the more complex an exercise is, the greater the possibility for trick action. After six months from the onset of the disease, resisted exercises should be instituted for all muscles that can overcome gravity. The patient should undertake practice exercise before assessing the maximum load of resistance the patient is capable of tackling. He must understand how essential is his contribution of mental and physical effort. The physiotherapist can aid by constant encouragement and by the judicious use of varied exercises to avoid monotony. There appears to be little doubt that progressive resistance exercises should be given to prime movers, and the best results have been obtained by simple movements.

Delorme and Watkins in their book *Progressive Resistance Exercises* suggest the following principles of treatment:

- (1) The patient is placed comfortably in the optimum position for the particular muscle requiring exercise
- (2) The maximum load assessment is registered in pounds by a spring scale whilst the patient exerts maximum effort.
- (3) Ten repetitions of the maximum load must be carried out in sets of three at every treatment, with rests of two minutes between each set of ten exercises.

Treatment is carried out daily for six months. A frequent re-assessment of maximum load must be carried out with the aim of increasing the load

- (4) The speed of the exercises should be approximately twenty-six per minute
- (5) The rhythm of work is set up with a metronome.
- (6) The muscle load must be sufficiently heavy to produce a sharp fatigue curve within $1\frac{1}{2}$ to 2 minutes

If the exercises produce fatigue and a diminished range of movement, they must be reduced in number and range. It is important to note, however, that the strength of a muscle may vary from day to day, owing to central factors such as

(2) FREE MOVEMENTS

In this form of treatment, no assistance is given by the physiotherapist. The movement may be carried out in the early stages in the treatment pool or in total suspension slings. Voluntary exercises may also be performed with the limb placed in supporting slings. By using these methods, friction and the force of gravity are eliminated and so various planes and axes of movement can be achieved. The dynamic splintage given by spring suspension simulates the water treatment and encourages normal muscle movement and rhythm.

Free movements are of great value in the later stages when co-ordination exercises are given in the lying, sitting and standing positions.

(3) RESISTED MOVEMENTS

This is undoubtedly the most important part of muscle re-education as the primary function is to strengthen the muscles. The remaining innervated muscle fibres appear to respond to progressive resistance exercises by an increase in their strength and work capacity, in practically the same manner as normal muscles. In the early stages, as in manual assistance, manual resistance is the best method of giving resistance exercises, because minute variations can be given and the resistance carefully graduated to the returning power of the muscle.

Resisted movements may also be given by spring suspension in which the limb to be treated is supported comfortably in slings suspended from an overhead frame and a spring of suitable tension introduced into the circuit. Later as the muscle function improves, more effective and progressive resisted exercises can be planned with the aid of springs of varying tension, or pulleys and weights. The advantage of the latter method is that the patient's progress is obvious to him, thus affording him great encouragement.

With the frequent muscle charting previously advocated, it can easily be assessed when any muscle group has improved sufficiently for the treatment to be "progressed," and an

attempt should be made to get the muscle to work at its maximum strength

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mental fatigue, or to local factors such as alteration in the temperature of a limb.

In our unit, active exercises with springs and slings, and active exercises in the Hubbard's tank are the two most important methods used.

Trick Movements

These may be due to an unstable fulcrum, or to a sudden relaxation of antagonists immediately prior to the patient attempting the desired movement. All muscles do not recover at the same rate so that the inco-ordinated movements can develop at any stage in the recovery. If exercises are not expertly controlled, the patient tends to use and develop the normal muscles, thus increasing the muscular imbalance and the tendency to deformity. The reaction of a weak muscle may entirely disappear when there is overactivity of stronger muscles in the area. The weakened muscle ceases to function because it tires readily, and if the patient continues to use the limb, a new pattern of movement develops. If the functioning muscles can be kept at their normal length, and if these inco-ordinated movements are not established, deformities can be prevented.

Four main theories have been advanced to explain the phenomenon of muscle inco-ordination:

- (1) Functional disturbances of the upper motor neurones
- (2) Abnormal functional activity of the phenomenon of reciprocal innervation.
- (3) Irregular destruction of the anterior horn cells.
- (4) Acetylcholine hypersensitivity.

COMMONER TYPES OF TRICK MOVEMENTS

- (1) Thumb movement is frequently limited by paralysis of the abductor pollicis brevis and opponens pollicis. Opposition of the fingers and thumb to form the normal round O can often be performed by the combined action of abductor pollicis longus and flexor pollicis brevis.
- (2) Similarly when flexor pollicis longus is paralysed, trick

flexion of the terminal phalanx can be performed by extending the wrist and abducting the thumb.

- (3) In weakness of the interossei, extensor digitorum longus, extensor indices and minimi digiti may abduct the fingers, and the fingers and hand should be exercised on a flat smooth surface when any weakness can readily be detected
- (4) The fingers can be flexed by the wrist extensors if there is some shortening of the long flexors.
- (5) The lateral abdominals will cause "hitching" of the pelvis on abduction, if there is weakness of the hip abductors on that side
- (6) Weak hamstrings can be substituted in knee flexion, by flexion of the hip if lying in the supine position.
- (7) Paralysis of the quadriceps can be substituted by the gluteus maximus contracting in extension of the knee.

If after a reasonable period of time, severe paresis or paralysis persists and recovery seems to have ceased, "trick" movements should be encouraged and the surviving muscles developed.

Fatigue

Great care must be taken by the physiotherapist throughout the treatment to appreciate the early onset of fatigue in any muscle or muscle group, and adequate periods of rest must be given.

Fatigue can be recognized in a muscle if the last contraction does not show the same degree of power as the first, or by a diminished range of movement during the immediate treatment or on the following day. Rest is then usually prescribed for two to three days

Fatigue is assumed to arise from the accumulation of lactic acid, due to an incomplete metabolism and a defective circulation. Rapid fatigue of recovered muscles appears to be a permanent feature, and may be due to axonal branching with overloading of the individual neurones. In the more complex activities, fatigue promotes asynergy and inco-ordination which interfere with the restoration of the normal

patterns of activity. Early activity is often curtailed because of the danger of fatigue producing further weakness, but the danger of inco-ordination produced by too strenuous exercises is probably a more important factor.

Recently it has been shown that deliberately induced physiological fatigue does no harm to the paretic muscle in the recovery stages of poliomyelitis.

Electrotherapy and Massage

ELECTROTHERAPY In 1892 Church advised electrical treatment *in order to maintain the nutrition of the paralysed muscles*, and in 1894 Jones stated that electricity acted only as a stimulating treatment and that it was superior to massage.

In recent times, electrical treatment has been used very extensively and its value and limitations are more fully appreciated.

The Faradic current produces a stimulus of short duration which is capable of obtaining an immediate response of all the fibres of a normal muscle, but is not able to obtain a response from a denervated muscle. The interrupted Galvanic current produces a stimulus which is of longer duration and a sluggish response is elicited from a denervated muscle. The muscle fibres lying immediately beneath the electrode respond, and the stimulus passes to the other fibres which respond with lesser rapidity the farther they are away from the point of stimulus.

The diagnostic value of Faradism can be useful in determining treatment. Affected muscles responding to a stimulus of short duration need no further electrical treatment. Muscle training in the form of graded activity must be employed to further recovery. Faradism carefully given is considered to be of real value in assisting muscle function and increasing muscle tone, but it is doubtful whether Faradic stimulation can compare in value with active exercises in muscle training. A point which cannot be overlooked is the possibility of fatigue through over-stimulation. The indiscriminate use of Faradism can certainly do more harm than good.

Interrupted Galvanism has been proved to be of value in maintaining the muscle tone of a paralysed muscle and in preventing its atrophy. This has been shown from the work done in peripheral nerve lesions. Galvanic stimulation in cases of poliomyelitis is daily treatment, the frequency of stimulations being ninety stimuli per minute for three minutes to each of the affected muscles. This daily electrical stimulation prevents atrophy.

In poliomyelitis it is not known whether axonal degeneration takes place, but it is definitely known that after peripheral nerve sutures, normal nerve regeneration takes place at the rate of 1 to 2 mm per day, and also in poliomyelitis the period in which all worthwhile recovery takes place is six months. If, therefore, recovery depended solely upon axonal regeneration, the more distal muscles could not recover in six months.

Most muscles which recover, retain throughout the electrical excitability characteristic of innervated muscle. Therefore those anterior horn cells which are capable of recovery are not usually damaged to such an extent as to cause axonal degeneration, and electrical stimulation is thus not required.

Observers who advocate electrical treatment state that it keeps the tissues in the optimum condition for the return of their nerve supply by maintaining its circulation and nutrition. It prevents some atrophy of the muscle and to a certain extent sustains morale. It is of value when a patient is unable to contract and relax individual muscles successfully by ordinary re-educative methods. If this method is combined with exercises one may get just that valuable extra ounce of power out of a permanently weakened muscle.

The sinusoidal current is a low frequency alternating current and the value it provides in cases of paralysis is its effect on the peripheral vascular circulation. A rhythmic contraction and relaxation of muscles can be obtained which relieves vaso-motor spasm.

Sinusoidal baths can be of great help in the chronic stages of poliomyelitis. Cold, cyanosed limbs often accompanied by chilblains, can be greatly relieved by a course of this treatment, but unfortunately the relief is only temporary.

Some orthopaedic surgeons have been very strong oppo-

nents of the use of electrical stimulation for the following reasons:

- (1) The time-consuming factor, if it is included in the routine treatment.
- (2) They have never been fully convinced of its value.

The latter is probably due to the fact that electrical stimulation has previously been used indiscriminately in all types of cases, whereas obviously the types which would respond best are those with a definite pattern of paralysis. Certain selected cases of paralysis of a single muscle or a single muscle group have been found to improve considerably with electrical treatment. This may be due to the new types of apparatus and the greater understanding of the physiotherapists

The square wave voltage stimulus which is produced from electronic generators gives a much more accurate method of determining the excitability of muscles and nerves.

Short Wave Diathermy

In the acute stage, diathermy has been said to reduce oedema of the cord and to promote the circulation by dilatation of the blood-vessels and so assist recovery of function, but this statement entirely lacks substantiation. It is also said to relieve partly or completely the acute pain in the muscles by causing muscular relaxation.

In the later stages, it is indeed possible that local heat improves the circulation in the paralysed limbs and so helps to maintain the nutrition of the muscles until active innervation is restored. Diathermy is also thought to be of value in relieving stiff and painful joints.

Clearly none of these procedures can have any influence upon the lesion in the damaged neurones and too much must not be expected of them.

Electrical stimulation of muscle is, in our opinion, of little value, but electrical stimulation of the phrenic nerve might prove the treatment of choice in respiratory disturbance of bulbar origin.

MASSAGE

It must first of all be stressed that this form of treatment should never be given except by a skilled physiotherapist.

In recent years massage has become of less importance in the field of physical medicine because it plays a passive role in treatment, whereas in modern treatment the accent is on activity. The need for the patient to contribute a mental and physical effort on his own behalf is now appreciated.

Massage can be given in some cases when constipation is due to paralysis of the abdominal muscles which are treated twice a day.

Some surgeons believe that massage is contra-indicated in the acute and early convalescent stages, whilst others believe that massage has very little real value in the treatment of poliomyelitis compared with active exercises in water, muscle re-education, etc. These points are not disputed, but massage must never be considered as a substitute for muscle re-education.

The value of massage in paralytic cases is to stimulate the circulation of blood and lymph, particularly in the distal parts of the limbs which are so often badly affected and so deprived of the muscular pump. If given gently, it can be very comforting to a patient with muscle and joint pain, and it helps to increase the muscle tone by improving the local nutrition. It also acts by retarding and counteracting muscle atrophy. If contractures are present, deep stretching frictions around the contracted tendons can also bring relief to the patient.

Careful massage to the weakened or paralysed muscles has also been advised after the temperature has been normal for one week. When the muscular pain and tenderness have disappeared massage in the form of effluage with very gentle kneading and friction around the joints may be given and later increased. This helps to maintain the blood supply to the paralysed limbs.

Muscle and joint pain can be relieved and deep friction around contracted tendons will aid treatment when contractures are present.

Local fatigue due to excessive reflex effort must be avoided, and the treatment periods should be of short duration—five

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(2) *Muscular Inco-ordination.* This is said to arise in the central nervous system and is involuntary. It is due to motor impulses intended for one muscle spreading to other muscles or muscle groups, producing abnormal patterns of motion, and to ineffectual contractions within the muscle itself instead of a co-ordinated rhythmic contraction. There is therefore a need for muscular rehabilitation.

(3) *Mental Alienation.* This is said to be an inability to produce a voluntary purposeful movement in an apparently normal flaccid muscle in spite of the fact that the nerve paths are intact, i.e. it is purely functional. This results when a muscle is pulled beyond its normal resting length by an opponent in spasm, when an attempt to contract the alienated muscle causes pain in its affected opponent and when changes in the nervous system interfere with normal neuro-muscular action. All alienated muscles are opposed by muscles in spasm.

The following are the essential points of this form of treatment. The patient is kept flat on a firm bed and a small towel is placed between the shoulders to prevent winging of the scapulae. The limbs are placed in their normal positions, the arms are placed alongside the body in the mid-horizontal position and folded towels prevent stretching of the muscles of the shoulder girdle. The forearms are placed midway between pronation and supination. The legs are placed together with slight flexion of the knees and the feet supported by a vertical fracture board.

Spasm and pain are treated by hot packs made of woollen cloth, and cut to fit the part of the limb to be treated. Large joints should not be included in these packs as free movement is to be encouraged. The packs may be applied at intervals varying from a quarter of an hour to two hours, from 7 a.m. to 7 p.m., and should be continued until the spasm is relieved. The alternate heating and cooling of the muscle is said to produce muscular relaxation and thus relieve pain and muscle tenderness. Certain workers attribute the weakness of movement to the muscular spasm of the antagonists and state that the paralysis of the affected muscles is due to mental alienation.

minutes only, but they may be repeated two or three times a day. Picking up or friction to the antagonist muscles may also be given.

It is only right to point out that local hyperaemia is more easily and safely attained by the use of warm baths and infra-red rays, whilst contrast bathing may also be useful.

When staffing a large unit for the treatment of poliomyelitis, it must be borne in mind that each patient requires a great amount of individual attention from the physiotherapist. It is obviously impossible for each physiotherapist to devote her whole time to a single patient, and therefore in determining which type of treatment to employ, preference must be given to that which in the opinion of the surgeon gives equally good results even if administered for a shorter time.

Sister Kenny Treatment

The section on treatment would not be complete without a few words on the greatly publicized Sister Kenny treatment. There is a most extensive American literature describing this method of treatment, but full details will not be given here as recent electromyographic and physiological studies show that it appears to have no scientific basis (Kenny, E., 1942 and 1944)

The terminology which Sister Kenny uses is as follows:

(1) *Muscular Spasm*. This is said to be present as the earliest symptom in all cases including those in the abortive and preparalytic stage. It includes fibrillary twitchings, hyper-irritability to stretching and a more or less involuntary tonic contraction and shortening of the muscle fibres which can often only be overcome by considerable force. The spasm affects chiefly the back and neck muscles, quadriceps, hamstrings, gastrocnemii, pectorals, biceps brachii and the muscles of respiration. This spasm more frequently affects a group of muscles than an individual one, and is usually accompanied by pain, irritability and increased tone. If it is untreated, atrophy, paralysis and subsequent contractures are responsible for the crippling after-effects.

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The affected limbs are moved passively several times a day through the greatest range which can be tolerated without producing pain. Active treatment should begin as soon as possible and should include muscle re-education. Massage and electrotherapy are not used. The patient should be maintained early in the normal standing position, but splints and braces should not be used. Mills (1938) suggested that deformity is caused by ischaemia, and that muscle spasm and fibrosis are accentuated by splinting.

Artificial respirators are not used. Detailed muscle charts are not kept as Sister Kenny objects to the early examination of muscles.

PSYCHOLOGICAL APPROACH

The patients and their relatives are encouraged to believe in the maximum possible recovery, and the bright mental outlook is stressed. Any residual paralysis is thought to be due to the late initiation of treatment or to the fact that it was not properly administered. It is stated that there is no spontaneous recovery or improvement.

Observers who have followed the Kenny treatment state that the duration of pain, tenderness and spasm is greatly shortened and that contractures and deformities are prevented.

Most careful objective attempts to assess the value of the Kenny treatment have repeatedly failed to support its claims, and there is no evidence that it can completely or even significantly prevent paralysis or deformities, nor do the results appear to differ materially from those obtained by other methods.

The report of the committee for the investigation of the Kenny treatment of poliomyelitis was published in 1944, and the main conclusions arrived at were as follows:

- (1) Lack of accurate records of reports of muscle examinations and so on, mitigate against scientific findings.
- (2) Miss Kenny's statement, that under "orthodox treatment" only 13% of the patients recovered without paralysis, whereas under her treatment over 80%

recovered, is believed to be a deliberate misrepresentation of the facts of treatment by other methods

- (3) Early Kenny treatment does not prevent paralysis; in some cases seen by the committee, there was actual progress of the paralysis after the Kenny treatment had been instituted.
- (4) The committee was of the opinion that there was questionable value in applying continuous hot packs to patients with minimal evidence of spasm.
- (5) Finally, the committee disapproves and condemns the wide publicity which has mislead the public and many members of the medical profession.

BIBLIOGRAPHY

- CHURCH, A. (1892) *Cincinnati Lancet-Cl*, 29, 519
 .. 13, 32
 .. 1st N Amer, 3, 12
 .. FOR INVESTIGATION,
 ..
 LOWMAN, C. L. (1932) *Amer J Nurs*, 32, 7
 LOWMAN, C. L. (1935) *Physiother Rev*, 15, 123
 MILLS, F. H. (1938) *Brit med J*, 1, 168

CHAPTER XIV

INTERMEDIATE STAGE

THIS stage commences with the disappearance of the muscle pain and tenderness and may last for two to three years, according to the severity and the extent of the paralysis, i.e. until the recovery of muscle power ceases, and this may be complete or only partial.

The aims of treatment must be to keep the joints of the paralysed limbs mobile and to produce hypertrophy in the remaining muscle fibres. In some cases, this will allow the patient to discard an appliance or give a better prognosis when muscle transplantation is indicated.

The affected limbs should be supported so that every joint is in the optimum position for the function of the limb.

Splints may be required as follows:

- (1) To keep the limb at maximum rest.
- (2) To oppose the influence of gravity.
- (3) To avoid undue stretching of a paralysed muscle beyond its normal resting length and therefore protect it.
- (4) To avoid shortening of healthy antagonists of the paralysed muscles.
- (5) To keep the joints in alignment.
- (6) To prevent deformities.

The simplest and lightest possible apparatus should be used at all times, as heavy appliances may do more harm than good. The splints may be made of aluminium or one of its alloys, one of the modern plastics or of light removable plaster of Paris. They should be well padded and great care should be taken to prevent pressure sores over the bony prominences. The splints should be removed at least twice a day, to allow the physiotherapist to carry out the treatment.

in a rigid splint, the following complications would be liable to occur

- (1) Atrophy and muscular weakness.
- (2) Loss of tension of the muscle.
- (3) Interference with the circulation of the muscle and the limb as a whole.
- (4) Peri-articular adhesions.
- (5) Atrophy of bone.
- (6) Loss of active and passive movement.
- (7) Inability to exercise the muscles effectively.

The type and degree of protection, however, depends on the following factors:

- (1) The degree and location of the muscular weakness
- (2) The amount of pain or muscle spasm.
- (3) The condition of the ligaments supporting the joints and the stability of the joints.
- (4) The relative strength of the opposing groups of muscles.
- (5) The duration of the disease since its onset

We find that individual prescribing is essential to determine whether a splint is required.

When activity is increased, care should be taken to use appliances to hold the weakened and paralysed part in a position so as to encourage co-ordinated action.

The protection by means of splints and appliances is usually necessary up to and during the intermediate period, or until the patient has made the maximum recovery.

In the later intermediate stage, further indications for the use of appliances are:

- (1) To mobilize the patient by supporting unstable limbs or other parts of the body.
- (2) To correct and prevent deformities and so avoid surgical procedures.
- (3) To protect, assist or substitute for weakened muscles.
- (4) To immobilize, mobilize or limit movement of joints.
- (5) To balance muscles and therefore maintain neutrality of muscle pull.

Let us now consider the various measures employed during the intermediate stage in the different regions of the body.

(1) Head and Neck

The head and neck should be held straight without flexion or extension and a cotton wool collar may be all that is necessary to support the neck muscles. Later, a Schrock type of brace or one of its modifications may be used.

(2) Abdominal Muscles

Gravity is the opponent of the spinal muscles. According to the position of the patient, different muscles act to produce the movement of the spine.

The muscles are attached to the ribs, whilst the intercostals, rectus abdominis and the obliques are prime movers of the spine.

In the erect position, gravity flexes the spine.

Flexion When the patient attempts to sit up from the supine position, with the arms folded, the recti and the obliques contract. In this way the abdominal muscles act on the spine through leverage of the ribs and pelvis. The rectus abdominis may be affected alone or along with the other flexors. These muscles are usually affected asymmetrically. In bilateral paresis the patient may not be able to raise his head and shoulders from the bed without raising his arms.

The tests for the abdominal muscles are:

- (1) Pressing the back downwards against the bed
- (2) Raising the head from the bed.
- (3) Holding the legs raised.
- (4) Long forced expiration (hissing).
- (5) Ballooning the abdomen.
- (6) Raising and turning to one side.
- (7) Lateral bending.

The lower intercostals are frequently affected with the abdominals. Deviation of the umbilicus or local bulging of the abdominal wall may be noticeable.

Extension The erector spinae extends the spine and is

divided into three columns, cervical, dorsal and lumbar. Any of these areas may be involved. In unilateral paralysis of the spine or where the erector spinae are unequally involved, scoliosis may result with convexity towards the weaker side. The erector spinae extends the spine and the para-vertebral cervical muscles, longus colli, longus capitis and the abdominals function in straightening the curves of the spine which is the first action in the cervical and lumbar region prior to forward bending which is carried out by gravity.

Lateral Flexion. The muscles involved in lateral flexion are the rectus abdominis, the obliques, the erector spinae, quadratus lumborum and the latissimus dorsi. In side flexion from the erect position the quadratus lumborum of the opposite side acts to check bending which is affected by gravity. To test the side flexors of the spine, place the patient in the side lying position. If there is any weakness, the patient cannot raise his shoulders from the bed whilst sliding his hand down the lateral aspect of his thigh.

Weakness of the anterior and lateral abdominal muscles is best treated by an abdominal belt with elastic insertions, as it is most important that the abdominal wall should not sag and become pendulous.

(3) Spine-Scoliosis

Mesener (1892) gave the first description of paralytic scoliosis following poliomyelitis, and Fortescue-Brickdale (1907) reported on a case with paralysis of the abdominal muscles and scoliosis. Desfosses (1909) studying this question, suggested that the atrophic bone changes might play a part in determining the direction of the scoliotic convexity.

Scoliosis can be one of the most rapidly progressive deformities due to muscle imbalance. Other factors which may possibly influence the severity of scoliosis are modification of the ligaments and alteration of the intravertebral discs with changes in the vertebrae. This condition may become permanent and when established is incurable except by operation.

Examination of the patient's muscle chart will immediately show the likelihood of scoliosis and provided that it has not already commenced, it may be possible to lessen its severity.

The prevention of deformity is a complicated and difficult problem because deformity may develop even in recumbency and may result from one or more of the following conditions:



FIG 14

(a) INABILITY TO SIT WITHOUT SUPPORT

(b) FITTED WITH TRUE SPINAL SUPPORT

[Reproduced by permission —Capener, III (1949), *Postgrad med J*, 28, 26]

(1) Involvement of part or whole of the intrinsic musculature of the spinal column. In the erect position the spine inclines away from the weak muscles

(2) Paralysis of the anterior and/or lateral abdominal muscles especially if unilateral.

(3) Paralysis of the psoas and gluteal muscles with loss of bulk in the region of the thigh and buttocks.

GENERAL MANAGEMENT

- (4) Paralysis of one or more limbs.
- (5) Paralysis of the shoulder muscles especially if this leads to an asymmetrical disuse and atrophy of the shoulder girdle.

With unilateral deltoid and biceps paralysis, a lateral curvature can be caused by attempts to raise the arm by tilting the trunk. The concavity is to the side of the paralysed deltoid.

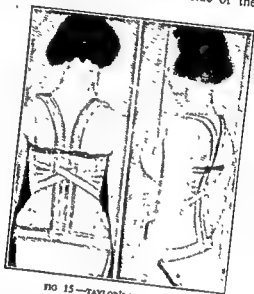


FIG. 15—TAYLOR'S SPINAL BRACE

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- (6) Shortening of one leg.
- (7) Contraction of the rectus femoris and ilio-tibial fascia producing a severe deviation of the lumbar spine towards the side of the contracture.
- (8) Unilateral paralysis or contracture of the quadratus lumborum, ilio-psoas and superficial erector spinae group.
- (9) Asymmetrical weakness of the sternomastoid group of muscles.
- (10) Change in length and resilience of the fibrous tissue supporting the spine and muscles.

Every effort should be made to develop the spinal muscles, but if scoliosis develops, the spine should be kept mobile, because this will give a much better result if a spinal fusion is required at a later date.

Structural deformities are due to the persistence of faulty alignment of bodily segments during rest and activity.

Scoliosis is controlled by early recognition and the care of the basic causative and accelerating factors.

If the spine or trunk muscles are involved, the correct bed

posture must be maintained, as this protects the abdominal and gluteal muscles. The patient should be kept supine in a plaster bed or spinal frame for six to eight months or even longer. The back should be kept straight and a restrainer should be used when necessary for children. When the patient is allowed up, the period of recumbency depending on the extent of the paralysis, he is fitted with a spinal support if there is any suggestion of spinal curvature

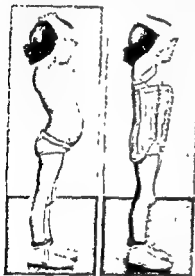


FIG 16 —SPENCER SPINAL SUPPORT

The curve in scoliosis is initially a postural one, but unguarded weight bearing results in a severe and extensive form of structural scoliosis.

A Green's corset is usually recommended for the support of the lower back, whilst scoliosis is said to be controlled by a Hoke's corset or by Bennett Kendall's, Fischer's or Taylor's brace fitted if necessary with a thoracic sling Kleinberg's spinal brace may also be most useful in cases which tend to develop lordosis or scoliosis. If both abdominal and spinal supports are needed, Goldthwait's or Bennett Kendall's type of spinal brace is required. In certain cases, these braces may

be used to correct deformity by exerting pressure. Subsequent treatment depends on the occupation of the patient and the degree of paralysis of the leg.

Rotation If the movement is to the left, the right sternomastoid, and the left splenius capitis, obliquus capitis with other deep muscles contract. The sternomastoid acts only during the last part of rotation. If the sternomastoids are affected unequally, the stronger should not be exercised.

It has been suggested that a certain amount of tightness can minimize deformity in the spine, whereas mobilization without regard for severe underlying weakness of the trunk muscles may result in a rapidly progressive scoliosis which may have been minimized by allowing the stiffness to remain.

(4) Pelvis

Unilateral weakness of the abdominal muscles and quadratus lumborum will allow the pelvis to be pulled up on the other side.

In cases with severe paralysis of the hip muscles, a moulded leather girdle attached to long calipers by hinges at the level of the hip joint gives some support.

If there is severe paralysis of the spine and hip muscles, the leg appliances are fitted with extensions to articulate with the spinal support, but the weight of this apparatus is a limiting factor in its use.

With recurrent dislocation of the hip due to paralysis of the gluteal muscles, the Bennett Kendall hip splint is used, as weight is transmitted through a hinged metal flange so that the hip does not receive the weight on walking.

(5) Lower Extremity

Flexion contractures of the hip joint caused by paralysis of the psoas, tensor fasciae latae or rectus femoris can be aggravated by prolonged sitting. Hip flexion contracture is always compensated for by lordosis and a protruding abdomen.

The standard equipment is the long or short leg caliper, with or without certain modifications. The choice of caliper

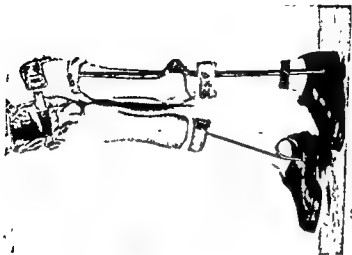


FIG. 19.—JOINTED CALIPER WITH POSTERIOR HINGE AND EXTERIOR TOE-RAISING COIL.
[Reproduced by permission—Capener, N (1949), *Postgrad med J*, 25, 23]

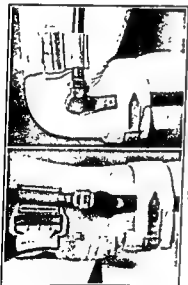


FIG. 17.—JOINTED KNEE CALIPER

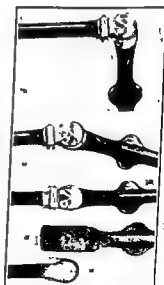


FIG. 18.—JOINTED CALIPER—LOCKING DEVICE
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depends upon the degree of paralysis, the age and the occupation of the patient. Various attachments such as



FIG. 20 —EXETER TOE-RAISING COIL SPLINT FOR DROP FOOT
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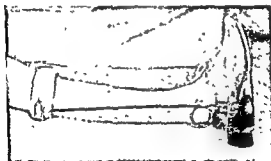


FIG. 21 —EXETER TOE-RAISING COIL ADAPTED AS BED-SPLINT
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T-straps, toe-raising springs, back-stops, spring locks at the knee, eccentric joints, bucket tops, pelvic bands, etc., may be ordered in individual cases.

The advantages of the long leg brace or Thomas's caliper are.

- (1) Its extreme simplicity and ease of manufacture.
- (2) It permits an early ambulant stage.

If the caliper is likely to be used for a long period, a locking joint at the knee should be provided, as this allows the patient to sit comfortably after a period of exercise.

If the thigh muscles are normal or only slightly affected, the use of a short caliper is all that is required to assist the involved muscles and to control the foot and ankle joint. In all cases when a walking caliper is used, it should be attached to the heel of the shoe by a heel tube which is welded to the sole plates. This heel tube must lie under the centre of the ankle joint and transversely to the line of the limb in walking. Round sockets permit normal dorsi- and plantar flexion, or a back-stop may be provided to the heel plate to prevent foot drop. Square sockets in the shoes prevent rotation, control moderate degrees of calcaneus and equinus, and hold the foot at right angles to the tibia.

If there is shortening of a limb of over one inch, the sole of the shoe must be raised, but full correction is unnecessary, because within the permissible limit of one inch, the pelvic tilt will compensate for this.

Flexion Contracture of the Hip with Weak Gluteus maximus
Unfortunately this may be missed for months especially if the patient has been allowed to sit up in the early stages and the joint has not been kept fully extended.

Mild cases may be treated by constant stretching of the contractures or by traction in a Thomas splint.

In more severe cases, the lordosis is first corrected by full flexion of the hip. The trunk and sound leg are then incorporated in a plaster spica with the hip and knee at right angles, and the affected leg is then pulled down by traction in a Thomas splint which is gradually lowered.

This deformity is encouraged by walking or by crawling before recovery is complete or if the patient walks without his appliance. If this is the only lesion, walking is possible with a compensatory lordosis. It may be associated, however,

with flexion contracture of the knees especially in children, and in these cases only quadruped locomotion is possible. There is also contraction of the tensor fasciae latae, sartorius, ilio-psoas, rectus femoris and the anterior capsule of the hip joint.

Ryerson (1933) reported on a case in which the quadrupedal form of locomotion was the result of a poliomyelitis contracture of the hip in flexion and abduction. Downward displacement of the tensor fasciae latae and gluteal muscles provided satisfactory correction, especially when it was followed by osteotomy of the upper portion of the femur.

Hough (1935) described five methods of correcting flexion deformity:

- (1) Stretching.
- (2) Subcutaneous tenotomy at the hip
- (3) Subcutaneous tenotomy of the ilio-tibial band
- (4) Open division of the ilio-tibial band
- (5) Fasciotomy at the hip.

Paralysis of the Hip Muscles In cases with gross hip paralysis, the best apparatus is the long double caliper with the lateral bar connected to a pelvic band by a bar carrying a hinge allowing full flexion, extension and 15° to 20° abduction and adduction. Such a control overcomes the loss of active rotation in walking.

Dislocation of the Hip The investigations of Elzinga and Key (1932) revealed that no definite type of paralysis is responsible for dislocation of the hip, but it is most common in hips with strong flexors, adductors and internal rotators, and weak extensors, abductors and external rotators. The abductors appear to be the most important in maintaining the stability of the hip joint. In dislocated hips, the acetabulum is shallow and severe coxa vara is usually present as the result of the paralysis. The deformities are the result of muscle imbalance and habitual faulty posture. If conservative treatment fails, the shelf operation is recommended.

Genu Recurvatum Minor degrees of genu recurvatum may be treated by raising the heel, but more marked degrees

The advantages of the long leg brace or Thomas's caliper are.

- (1) Its extreme simplicity and ease of manufacture.
- (2) It permits an early ambulant stage.

If the caliper is likely to be used for a long period, a locking joint at the knee should be provided, as this allows the patient to sit comfortably after a period of exercise.

If the thigh muscles are normal or only slightly affected, the use of a short caliper is all that is required to assist the involved muscles and to control the foot and ankle joint. In all cases when a walking caliper is used, it should be attached to the heel of the shoe by a heel tube which is welded to the sole plates. This heel tube must lie under the centre of the ankle joint and transversely to the line of the limb in walking. Round sockets permit normal dorsi- and plantar flexion, or a back-stop may be provided to the heel plate to prevent foot drop. Square sockets in the shoes prevent rotation, control moderate degrees of calcaneus and equinus, and hold the foot at right angles to the tibia.

If there is shortening of a limb of over one inch, the sole of the shoe must be raised, but full correction is unnecessary, because within the permissible limit of one inch, the pelvic tilt will compensate for this.

Flexion Contracture of the Hip with Weak Gluteus maximus
Unfortunately this may be missed for months especially if the patient has been allowed to sit up in the early stages and the joint has not been kept fully extended.

Mild cases may be treated by constant stretching of the contractures or by traction in a Thomas splint.

In more severe cases, the lordosis is first corrected by full flexion of the hip. The trunk and sound leg are then incorporated in a plaster spica with the hip and knee at right angles, and the affected leg is then pulled down by traction in a Thomas splint which is gradually lowered.

This deformity is encouraged by walking or by crawling before recovery is complete or if the patient walks without his appliance. If this is the only lesion, walking is possible with a compensatory lordosis. It may be associated, however,

Calipers should be worn during the day, and in bed night splints are employed to avert foot drop.

patient wears an outside iron with an inner T-strap.

(6) Upper Limb

The standard type of splint used for paralysis of the abductor muscles of the shoulders is the Littler-Jones



FIG 22 —LITTLER-JONES ABDUCTION SPLINT

[Reproduced by permission—Capener, ■ (1949), *Postgrad med J*, 25, 24]

abduction splint or one of its modifications. If paralysis of a permanent nature becomes obvious, the splint should be discarded and treatment concentrated on the function of the elbow and hand. Neglect may allow adduction and inversion contracture at the shoulder from the pull of the pectoralis major and latissimus dorsi muscles. This must be corrected by gradual stretching under anaesthesia, or by tenotomy.

The paralysed biceps is relaxed throughout recovery in a plaster of Paris gutter splint, angled at 90° , with the forearm in full supination.

In paralytic lesions involving the forearm and hand, the

should be controlled by a knee cage or caliper, whilst the severest cases require operation.

Paralysis Below the Knee. Every case with paralysis below the knee can be treated by operation, and the appliances discarded. The indications for operation, however, depend partly upon whether the residual paralysis above the knee still requires the use of an appliance. Any muscle, group of muscles or combination of groups of muscles may be affected, and the following are given as examples with the equivalent deformity.

<i>Deformity</i>	<i>Muscles Affected by Paresis or Paralysis</i>
(1) Talipes equinus.	Dorsiflexors of the foot.
(2) Talipes equinovarus.	Dorsiflexors of the foot and the peronei. The action of flexor hallucis longus and flexor digitorum longus are unimpaired.
(3) Talipes varus.	Peroneus longus and brevis.
(4) Talipes valgus.	Tibialis anticus and/or posticus. Flexor hallucis longus. Weakness or complete paralysis of the whole leg.
(5) Talipes equinovalgus.	Dorsiflexors and invertors of the foot.
(6) Talipes calcaneus.	Muscles of the calf.
(7) Talipes calcaneovalgus.	Muscles of the calf with tibialis anticus and/or posticus.

It is important always to remember that a non-paralytic talipes equinus will develop if the foot is allowed to drop under the weight of the bedclothes.

For mild degrees of deformity, the shoe should be modified as follows:

Talipes valgus	— Raise inner border of shoe.
Talipes varus	— Raise outer border of shoe.
Talipes calcaneus	— Raise the heel
Talipes equinus	— Shoe should have a flat heel.

Finally, it is most important to recognize that orthopaedic appliances may be used as a temporary expedient or as a permanent feature in the treatment of poliomyelitis. In the former case, these appliances may be used until the affected muscle or muscles have regained sufficient power to prevent unnatural positions, over-stretching of the paralysed muscles, until a decision to operate has been reached or the patient is old enough for operation.

RE-EDUCATION OF POSTURE AND WALKING

Posture and Balance

Even though the patient is unable to use his arms, the trunk and the legs must be trained to maintain balance. The trunk muscles must be paid to the triceps, flexors and extensors of the wrist, and the movements of the fingers. Even with extensive flaccid paralysis of the leg, abdominal and back muscles, without flexion contraction deformities of the hip, knee or ankle, or if these have first been corrected, and the patient's arms are capable of using crutches, he can be taught to balance correctly and later to walk.

Postural balance is maintained by the postural reflexes. These are activated through the proprioceptive system in the muscles and joints, the eyes and the labyrinth. These reflexes are outside the control of volition and keep the body in the erect position.

The erect position is maintained by every joint being balanced tonically. The supporting muscles which are working against gravity pull harder than those of the opposite side. Thus the tonic contraction of muscles controlling joints is increased when balance is altered by gravity. A good posture is one in which the minimum of muscle activity is required in controlling the erect position. A poor posture requires increased effort to regain a good position, and in doing so, fatigue quickly results.

Balance, both local and general, is disturbed when there is paralysis of a muscle, disability being caused more often from deformity through the disturbed balance than from

emphasis is on mobility because this helps to prevent contractures and joint stiffness. This principle is well illustrated in the splints devised by Brian Thomas (1944), Bunnell (1946), Napier (1946) and Capener (1946).

If the hand is useless, a permanent splint should be worn.

The orthopaedic surgeon is familiar with the various types of appliances and the indications for their use, and he will ascertain regularly that they fit accurately and support the affected part. He will decide upon the length of time they are to be worn, bearing in mind that frequent observations and adjustments are essential in children. In addition the



FIG 23—BRIAN THOMAS EXTENSOR APPLIANCE

[Reproduced by permission—Capener, N (1949), *Postgrad med J*, III 24]

patient is given specific instructions regarding the apparatus, when it is to be worn, the amount of activity allowed, etc.

In specific cases, the mother may be taught to remove and re-apply the splint. This is essential in small children, as the appliance will require cleansing when soiled. Absolute cleanliness is essential in all cases where appliances are used for the mechanical assistance in stance, locomotion or to correct deformity.

It is a great advantage if the necessary splints can be produced in the hospital workshop under the direct control of the orthopaedic surgeon. Under the National Health Service, all appliances are supplied free of charge, including motor and electrically propelled chairs with the controls modified where necessary for the more severely paralysed patients.

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Balance, both local and general, is disturbed when there is paralysis of a muscle, disability being caused more often from deformity through the disturbed balance than from

paralysis. Both contractures and paralysis of muscles may cause ligaments to stretch, these being subject to continual strain. The stretching of the ligaments may cause subluxation. Unexpected deformities occur due to the action of gravity, e.g. hyperextension of the knee due to attempts to lock it. Once deformities are established, they progress rapidly even though they commence insidiously.

Principles of Re-education for Posture

- (1) Restore and maintain mobility of the joints.
- (2) Correct bad habits.
- (3) Develop muscle sense by relaxation.
- (4) Re-establish co-ordination and control.

Treatment

In poliomyelitis, the continual effort of adjustment in balancing against gravity induces fatigue and in a programme of treatment rest must have an essential place.

Posture training must begin in the lying position and only progress to sitting and standing when the postural sense is achieved. Corrective posture exercises of breathing and balancing must be taught in the lying position, progressing to sitting and finally standing.

Joints which have a tendency to stiffness should be treated by manipulation and passive movements frequently. If this is done from an early stage in the disease, a full range of movement can be obtained. The small joints of the hands and feet and spine are very difficult to free once stiffness has been established.

Structural modifications of the ligaments and capsules of joints and in the spine, changes in the inter-vertebral discs may become permanent.

Mobility exercises must not be given to the spine if there is a likelihood of scoliosis because the presence of a degree of tightness can minimize deformity.

Faulty attitudes brought about by disability must be corrected. These can occur at an early stage of the disease whilst the patient is still recumbent. A child with paralysis

of the lower limbs may be allowed to sit up frequently and this may cause contraction of the hip flexors and stretching of the gluteus maximus.

The position of a child's bed, if not frequently altered, may be the cause of scoliosis. If the bed is against a wall, the child will turn in one direction, and constantly lifting the head may lead to an aggravated cervical curve.

Relaxation forms an essential part in corrective training; also breathing exercises with the trunk held habitually in good posture. Natural breathing with full use of the diaphragm must be the aim.

Exercises directed towards re-educating the postural reflexes in co-ordination and control must be commenced in the horizontal position. The gravity-resisting muscles such as the intrinsic of the foot, quadriceps, glutei, trapezius and pre-vertebral cervicals must be exercised individually.

When the patient has learnt control of these muscles, exercises must be given to co-ordinate action by the glutei and the abdominals working together to flatten the lumbar spine. The mastering of co-ordination can only be successful if gravity is eliminated.

Every patient should have postural correction because any weakness of the muscles of the upper limb, lower limb or pelvis may affect the position of the head, the upper part of the trunk or the whole body posture. The correct posture should be taught at the earliest possible moment, even while the patient is in the supine position and he must attempt to maintain the corrected position voluntarily.

In cases with isolated upper limb paralysis, the patient can be placed in the sitting position at an early date, but no definite time can be given because here as in other cases to be described later, this will depend upon the rate of progress and his response to treatment.

bency The best guide to the appropriate time is when the muscle chart shows no further improvement.

If the spine, neck, trunk and shoulder girdle muscles are

markedly involved, sitting up may not be possible for twelve to eighteen months. The patient may even then require the aid of a spinal support, otherwise scoliosis and kyphosis may develop rapidly and become permanent. Sitting posture is assumed very gradually indeed, and in the beginning it may be allowed for only a few seconds once or twice a week. When the patient is accustomed to sitting up, he should begin balance exercises by the use of movement of the head, arms and legs. Later, flexion, extension, lateral flexion and rotation of the trunk are introduced, and any tendency to scoliosis is watched for very carefully. If this is observed, mobilizing and hyperextension exercises should be given.

If the back, gluteal and leg muscles are extensively involved, weight bearing was formerly postponed for approximately eighteen months, but it may be begun much earlier in a treatment pool which is fitted with parallel bars. Provided the patient's arms are strong enough, it is possible to teach balance and weight bearing in water approximately four to six months from the time of the initial attack.

When the patient is first allowed out of bed, great care must be taken to avoid general fatigue. First of all he must concentrate on co-ordination of his muscles and on balance. Standing balance should be practised and well developed before walking is attempted, otherwise a most unstable gait may result. Careful observation is required to detect any tendency to deformity, particularly of the back and pelvis. A patient wearing an arm abduction splint must be watched carefully and, if necessary, postural exercises are prescribed.

When the patient starts to walk, instruction is given in foot placing, co-ordination of the arms and legs, shifting of the body weight, and such control of various regions of the body as the individual patient requires. Parallel bars and walking machines are excellent for this purpose, but again any erroneous deflection of the body weight during walking must be prevented. Crutches and walking-sticks can also be used when required, but they may always be necessary to allow the patient to get about. The patient should be taught to take steps of appropriate length and he may be instructed in "four-point" walking, or in the use of tripod gait.

Experience has shown that quite a number of poliomyelitis patients have been unable to master the technique of the tripod gait and this is possibly due to the weakness of the muscles of the trunk and upper extremities

The patients should be taught to get in and out of bed unaided, and he may even learn with patience to sit down on, and get up from, a chair or lavatory seat. Even a very severely paralysed patient should be able to ascend and descend stairs.

Although the patient may have shown no apparent improvement for several months, he may after assuming the erect posture, progress to a re-

markable degree. On the other hand, loss of the power of the muscles may occur. This is due to over-fatigue or from a too early removal of the supports. Unfortunately in a few instances this damage may be permanent.

Summary of Postural Training

General activity exercises must not be given because they increase the faulty posture through fatigue.

The patient must be taught correct posture with the aid of a wall mirror.

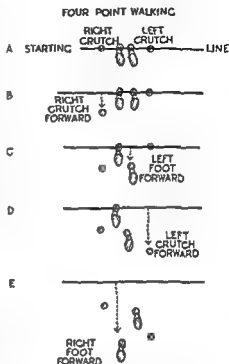


FIG 24

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weak, it is sometimes necessary to train the adductor magnus which is a weak extensor of the hip. This can be achieved with the leg internally rotated.

Quadriceps—if the quadriceps muscle is paralysed, the patient usually has to wear a caliper. Its true function is as a fixator of the knee and extension is secondary to this.

Calf—if both plantar flexors and dorsiflexors are involved, one should aim for the recovery of plantar flexion.

Class Exercises and Games

A number of patients can be given a class of free movement and light games. By this means the patients improve in general health and morale. They are not allowed, however, to join class exercises for the first six months of the disease as fatigue may prove detrimental to recovery. Exercises aim at developing powers of endurance, whilst the selection of exercises is carefully planned and consideration is given to the varying degree of paralysis of each patient.

The class is best conducted in a gymnasium with each patient lying on a mattress so that adequate rest can be obtained between exercises. Special class exercises should be given for the trunk, back and limbs, and competitive games are introduced for co-ordinated movements.

There are three class grades

- (1) Mat exercises—patient lying
- (2) Exercises mainly in sitting position
- (3) Activity in the erect position

Head exercises, trunk flexion, extension, lateral flexion and rotation, and limb and breathing exercises with frequent short rest periods are given.

The simplest apparatus may be used, e.g. balls of varying weight.

Organized games in the form of sitting P.T., basket ball, and volley ball are also useful forms of exercise. Archery has been found to be very beneficial for cases with upper limb palsies and wheel chair polo for paraplegic cases.

Posture and balance must be taught in the lying position.

Individual muscle action must first be controlled and then co-ordinated movements. When this is achieved, gravity can be introduced with the sitting position and finally the standing position.

A practical point which is well worth mentioning is how to measure a patient for crutches or walking-sticks. For long crutches, the measurement is taken from one inch below the axilla to the heel, whilst the patient is standing, or lying in the supine position in bed. Elbow crutches or walking-sticks should be measured from the level of the great trochanter to the heel.

Essential Muscles for Function

The two factors which require immediate attention are:

- (1) Recognition of the most important muscle for recovery.
- (2) The techniques of treatment which will aid recovery.

The following list of muscles may act as a guide to the physiotherapist considering priority of their importance.

Upper Limb. The importance of the muscles diminishes from the fingers to the shoulders.

Opponens—paralysis may occur with loss of opposition to the thumb and loss of skill in hand movements.

Flexors of the elbow—paralysis or weakness in the elbow flexors is a greater disability than loss of triceps. It must, however, be remembered that the triceps is an important muscle if the patient has to walk with the aid of crutches. A triceps grip added to a crutch can support the elbow if the triceps is weak.

Deltoid—the importance of aiding recovery to the deltoid for abduction of the arm is obvious.

The abdominals—the abdominal muscles function in respiration, control obliquity, and function in movements of the spine.

Lower Limb. The importance of the muscles diminishes from the hip to the toes.

Extensors of the hip—these are essential for walking. If the gluteus maximus is completely paralysed or is very

CHAPTER XV

RESIDUAL STAGE

WHEN this stage has been reached in about eighteen to twenty-four months from the onset of the disease, and provided that the patient has had correct treatment from the beginning, it may be found that protection is required indefinitely for the lower extremities or the back, to prevent a progressive stretching of the ligaments, and joint instability. Although 70% of all cases make a complete recovery without treatment, it is the seriously paralysed cases with imbalance of the opposing groups of muscles which require this protection for several years, if not for the duration of their lives.

In untreated cases, an attempt should first of all be made to correct the deformities by a period of conservative treatment with physiotherapy and mechanical fixation, before any operative treatment is undertaken

OPERATIVE SURGERY

A careful preoperative examination of the heart, lungs, urine, etc., should be made and any abnormality noted and treated appropriately. The operation area should be prepared for at least forty-eight hours before operation, and the type of preparation should depend upon the preference of the surgeon. Plenty of fluids with glucose should be given and if the patient is very dehydrated, intravenous saline with glucose 5% may be administered. It may also be advisable to give a sedative the evening before operation in order to ensure a good night's rest.

To avoid a later retardation of growth, all operations are preferably postponed until the child is ten to twelve years of age. It is better for a brace to be worn during the period of growth and surgery postponed than to perform a premature operation.

A number of orthopaedic surgeons are strongly of the opinion that all major operations should be postponed until

within twelve months of the acute attack if they are considered necessary. They should be performed if the offending contractures prevent the functional use of the opposing muscle groups, or disrupt the normal weight-bearing lines. This is especially true in cases with contractures about the hip joint and they should be treated early, because a persistent contracture of the hip abductors or flexors will shift the pelvis in a lateral or antero-posterior plane, in its relationship to the trunk and lower extremities. This would result in scoliosis or lordosis, and a disruption of the weight-bearing lines. These secondary deformities are found to be much more resistant to treatment than the original localized contracture of the hip.

If the joints are contracted, the shortened capsule and muscles may be stretched gently by simple types of traction or by a series of plasters, but if stability is essential as in the lower limbs or spine, then the joint should be arthrodesed. The range of movement so lost is usually compensated by an additional range of movement in other regions. In the upper limb, on the other hand, tendon transplantation is often used in an attempt to restore the accurate movements of the fingers.

Deformed bones may be straightened in adults by osteotomy, but the deficient blood supply and calcification of the bone in the affected limb may lead to a delayed union.

(A) TENDON TRANSPLANTATION AND TRANSPOSITION

Tendon transplantation which was first described by Goldthwait in 1896 and later by Tubby in 1901, has been largely abandoned as the main treatment in lower limb paralysis.

Great care must be taken in the selection of suitable cases for this operation, and the underlying pattern of paralysis must be the guide as to which muscles should be used to obtain the maximum function and support.

In general, the purpose of these operations has been stated to be

(1) To substitute the power of an active muscle for that

all hope of further recovery in the function of the part has been abandoned. This is usually at the end of about two years, but we are nevertheless of the opinion that this is much too long to wait in all cases. To mention only one example, earlier operation is definitely indicated when the deformity is increasing despite conservative treatment.

Several factors, all of which must be borne in mind, could produce the disability present, and thus the individual requirements of the patient may necessitate modifications of the operation contemplated.

The operations should be performed only in special centres, and by surgeons who are prepared to master the complicated technique required.

It is not possible to give full details of the operations, but I trust that the following résumé will be helpful, instructive and stimulating to the reader.

The indications for operation are, generally speaking:

- (1) To improve the muscle function and balance, by making effective use of the remaining muscle power.
- (2) To improve static stability, by making unstable movable joints stable and immobile.
- (3) To correct deformities.
- (4) To enable the patient to discard appliances.

It is, however, important to note that the surgeon must plan an operation or a combination of operations to suit each individual case, and in order to do this satisfactorily he must have a detailed knowledge of body mechanics.

Although forcible manipulation under anaesthesia is one of the simplest methods of correcting deformities, it is not without danger, because a fracture of the decalcified bone may occur before the shortened inelastic muscle or capsule yields.

If muscle transplants or arthrodesis are considered necessary, they should be deferred until full growth has been attained. Operations for joint stabilization should not be considered until a thorough attempt has been made to obtain maximum improvement.

Surgical correction of contractures may be carried out

because overstretching of active muscle will eventually lead to its paralysis, whilst if it is slack it will not pull with its full force

- (5) The nerve and blood supply to the muscle must not be impaired or injured
- (6) After operation, the limb must be elevated, and the joint fixed in an over-corrected position, i.e. the origin and new insertion of the muscle are approximated for about three weeks.

Early post-operative active and passive movements within narrow limits are allowed, and they are gradually increased in range, strength and power. The speed of the desired movements is developed and also the full functional adaptation into integral movements

A careful watch is kept for years afterwards for substitution movements and wrong habits, because transplantation not only transfers power to the paralysed muscles, but the power of the opponents is weakened

There is no doubt that if these operations have been carried out satisfactorily, the muscle of the transplanted tendon hypertrophies. Occasionally some of the power of the paralysed muscles recovers after re-education of a transplant and the summation of the power of the two muscles is sometimes equal to that of the original muscles before the onset of the paralysis.

Ober (1935) stated that tendon transplant was indicated in paralysis of the upper limb when there was impairment of opposition of the thumb, extension of the wrist, flexion of the elbow, and when it was necessary to prevent subluxation of the shoulder

Tendon transplant may precede a bone block operation to limit movements and to prevent over-stretching of the transferred tendons

Stabilization of the foot is also essential and knock-knee or bow legs should also be corrected

Other observers point out that in the forearm and foot, tendon transplants tend to precede arthrodesis when the operation has been done in two stages, because it was found

of a paralysed one, but in our opinion, this is the main factor which has brought tendon transplantation into disrepute.

- (2) To restore muscle balance and stabilize a part of the body.
- (3) To improve the appearance of a part, by preventing or correcting deformity.

The minimum requirements which must be fulfilled before performing these operations are:

- (a) Any deformity must be corrected, and there must be no deformity of the joint or joints to be acted upon by the transplanted tendon.
- (b) There must be a free range of passive movement.
- (c) The muscle of the transplanted tendon must be of adequate power, and able to be spared from its original action.

In addition to the above, certain principles must be observed before undertaking a tendon transplantation and these are,

- (1) A very careful selection of the tendon to be transplanted is essential, and it must be from healthy muscle and of a much stronger grade than the one which it is replacing. It must be capable of being trained in its new function, and should replace the action of the paralysed muscle or muscle group.
- (2) The site of implantation must be ideally chosen and the tendon must be fixed firmly and accurately into bone in the limbs, and also into tendon in the upper limb. Muscular, periosteal and fascial attachments in that order of preference may also be used but they are less satisfactory.
- (3) The route preferably straight or oblique to be traversed by the tendon is determined, and it usually tunnels in the subcutaneous fatty tissues or preferably in a tendon sheath. This is to try to avoid friction and the formation of adhesions.
- (4) The correct tension of the transplant is most important,

curve below, to that above, and centre it on the vertebra immediately between the two main curves.

The operative treatment for scoliosis is carried out principally in adults, and the main indications in special cases are as follows

- (1) The deformity cannot be corrected by conservative means
- (2) The deformity recurs when the treatment is stopped
- (3) There is a progressive increase in the curvature.
- (4) *Heavy appliances are otherwise necessary for the duration of the patient's life* Fusion is especially helpful if the patient also requires heavy leg appliances

Preoperatively, the deformity should be corrected as much as possible by the use of a Whitman's frame or by traction with a Sayre's halter. This correction is controlled by radiographs and it is usually maximum in four to eight weeks.

In adults, with an obvious weakness of the spinal muscles, fusion of the vertebrae should be carried out before walking takes place, and it may be preferable to do this in two or three stages.

With established contracted scoliosis, the curvature is corrected by a turnbuckle plaster and then the spine is fused in the position of maximum correction. The number of vertebrae to be fused is determined by the curvature. The optimum age for this operation is 14 to 16 years.

The operations usually advocated are

- (a) Hibbs's operation, with or without a bone graft from the tibia
- (b) Albee's operation
- (c) Kleinberg's operation

Postoperatively some surgeons prefer their cases to be nursed in a plaster bed, and then later wear a plaster jacket for a period of 12 to 18 months. Other surgeons obtain the maximum correction by means of Risser jackets. When the maximum correction has been obtained, a small window is cut in the jacket over the operation site and the graft performed. The window is then sealed with more plaster and

that when fixation of the joint was done first, the immobilization caused wasting and weakness of the muscles due to be transplanted.

(B) TENODESIS

This operation is not carried out in growing children, as it is apt to produce deformity. It should be reserved mainly for adults and adolescents with foot-drop who do not wish to wear a brace but who have a good stable ankle joint.

(C) OPERATIONS ON THE SPINE

Patients should be watched for several years where there has been trunk paresis. The degree of deformity in the adult never reaches the extremes seen in children.

During adolescence when growth of the trunk is accelerated, scoliosis and lateral curvature of the spine may develop rapidly and increase in severity to produce marked deformity. Usually there is no noticeable increase in curvature after sixteen years of age.

The primary curve occurs as the result of the imbalanced forces and may be single or double.

The compensatory curves develop in opposite directions above and below the primary area. They arise as the result of a tightening reflex in an attempt by the body to maintain the trunk in an erect and balanced position.

It is hardly necessary to point out that the majority of poliomyelitis patients who require bone grafting or fascial transplants to the spine, are poor subjects for operation. Chest complications are frequent and great care should be taken to avoid them. If really good results are to be obtained, the most careful selection of patients is essential.

If the general axis of the spine is faulty, it is unreasonable to graft a limited region, because this will gradually yield under the stress and strains which were and still are the cause of the deformity.

If the occipital foramen lies over the gluteal cleft, then even a number of spinal curves may be less disabling than a single "C" curve. One should graft across from the concavity of the

Intra-articular arthrodesis or an extra-articular method using Albee's, Trumble's or Brittain's technique can be used. The hip is fused in slight abduction, extension and lateral rotation.

Arthrodesis is contra-indicated in the presence of rigidity of the lumbar spine, and paralysis of the trunk.

Dislocation of the Hip

Complete or incomplete dislocation of the hip backwards and upwards follows flexion deformity associated with adduction, and it is also facilitated by the relaxation of the joint capsule. It adds instability to the limb and the head may be easily reducible in and out of the acetabulum.

Flexion and adduction should be prevented by conservative methods, e.g. by the use of an abduction splint like a Putti's mattress, but where there is recurrent dislocation, Albee's roofing operation with reefing of the capsule should be performed.

definite structural stability, they do not improve the limp which is due to muscle paralysis. Fusion not only provides complete stability, but also a great improvement in the gait. Paralysis of the quadriceps is not a contra-indication to operation, but arthrodesis of the hip must be done only if there is a good ligamentous integrity of the knee. The optimum position for an arthrodesed hip is 30° flexion, neutral rotation and slight abduction.

Hip Flexion Contracture

This results from contraction of the tensor fasciae latae, the ilio-psoas, sartorius, gluteus medius, rectus femoris, and the anterior part of the capsule of the hip joint. It is frequently associated with abduction of the hip, flexion deformity of the knee and talipes equinus of the foot. It is often overlooked until well advanced, and occurs if the joint is not kept extended, or if the patient is allowed to sit up in the early

subsequent operations are performed through other windows as required.

In this way, the correction is maintained throughout the period of operation and any slipping which might occur during the changing of the plaster jacket is avoided.

Fascial transplants have also been used in paralytic scoliosis, and for the correction of spinal curvature with depression of the affected shoulder.

Operations in Cases with Marked Weakness of the Abdominal Muscles

In cases with marked weakness of the anterior and lateral abdominal muscles, subcutaneous fascial grafts radiating from the umbilicus to the costal margins or iliac crests, may be employed after the patient becomes ambulant.

After the abdominal wall has been reinforced by Lowman's operation, the following advantages have been recorded:

- (1) Fatigue is less marked and there is an improvement in general physical well-being.
- (2) Lordosis is decreased.
- (3) Protuberance of the abdominal wall is less marked.
- (4) Sitting and walking are improved due to improvement in stabilization of the trunk on the pelvis
- (5) Control of the bladder and bowel is improved in some cases.

(D) OPERATIONS ON THE LOWER LIMB

Paralytic Pelvic Obliquity

Girard (1943) described an operation to correct this, by transplanting the origin of the hamstrings into the symphysis pubis.

Irwin (1947) advised subtrochanteric osteotomy in cases which could not be corrected

Flail Hip Joint

Instability and pain are best treated by arthrodesis in unilateral cases, especially if the knee is under control.

paralysed gluteal muscles by the tensor fasciae latae, the origin of which is transplanted with a portion of its bony attachment to a groove on the crest of the ilium near to the posterior superior spine. The action of the muscle is therefore changed from that of an abductor and a flexor, to that of an adductor and an extensor.

In Ober's operation, extension of the hip is obtained by the action of the sacrospinalis muscle on the femur.

It should be mentioned that muscle transplants for gluteus maximus paralysis are rarely satisfactory.

Paralysis of the Gluteus Medius

The limp cannot be compensated for by any apparatus or by building up the sole of the boot. Also no operation is indicated if this condition is accompanied by a marked involvement of the gluteus maximus.

Legg's operation appears to give the best chance of success. He recommended transplantation of the tensor fasciae femoris into the weakened gluteus medius, as he maintained that this diminished the lateral sway of the body and may eliminate the abductor limp.

Irwin (1947) advocates a subtrochanteric osteotomy for severe unilateral weakness of the gluteus medius, when the patient cannot otherwise be made ambulatory.

Paralysis of the Quadriceps extensor

Flexion deformity of the knee may result, with contraction of the hamstring muscles and posterior capsule of the knee joint.

Established flexion deformity usually yields to one of the following methods of gradual correction.

- (1) Traction in a Thomas splint
- (2) Successive wedgings in plaster of Paris.
- (3) Plaster of Paris with a turnbuckle behind the knee.
- (4) Tendon lengthening

Rapid correction has the risk of backward subluxation of the tibial head and sciatic palsy.

months It is also encouraged by walking or crawling before full recovery has taken place or without adequate protective apparatus.

The gait is characteristic and consists of a short step on the unaffected side with twisting of the pelvis.

In the earliest stages of the disease, mild cases may be partly obviated by placing the patient in the prone position for some hours daily, or by traction in a Thomas splint. Stretching is said to be *unreliable because of the local pain and irritation produced*. Also other tissues may be damaged and the circulation impaired. In severe cases, stretching is permissible by the Agnes Hunt method.

If operation is indicated, the tensor fasciae latae is divided and subperiosteal erosion of the soft tissues and muscles from the anterior superior spine and lateral surface of the ilium down to the anterior inferior spine is performed. If the anterior superior spine is too prominent, it may also be removed Postoperatively, gradual correction is carried out in a Schwartz frame or the patient is nursed in a plaster spica with the hips hyperextended for two to three weeks, so that the muscles may become united to their new attachments

In the more severe cases, Soutter divides the ilio-psoas muscle and the anterior portion of the capsule of the hip joint

Campbell's operation is reserved for the most severe cases. Here the anterior superior spine and the anterior portion of the iliac crest are detached along with their attached muscles, displaced distally, and reinserted at a lower level.

Contraction of the Tensor Fasciae Femoris

This is best corrected by Young's fasciotomy, and if treated early, deformity of the pelvis is prevented

Paralysis of the Gluteus Maximus

This may be unilateral or bilateral, and it can be associated

Arthrodesis can of course cure this deformity, but it is rarely necessary.

Genu Valgum This results from paralysis of the quadriceps femoris, and may be due to the stronger pull of the lateral hamstring muscles and particularly that of the tensor fasciae latae. The leg is also externally rotated and there is often a posterior subluxation.

If the angle is greater than 8° , a supracondylar osteotomy by MacEwen's method is indicated. In mild cases, treatment is by manipulation and plaster, and recurrence is prevented by applying a caliper fitted with a knock-knee strap to secure leverage.

Genu Varum. This is best corrected by removing a wedge of bone from the lateral aspect of the femur.

Flail Knee Joint

Arthrodesis of the joint should be performed by one of the established methods for a completely flail joint with irrecoverable paralysis of the quadriceps extensor muscle, if appliances could otherwise be discarded.

These established methods are

- (1) Intra-articular arthrodesis.
- (2) Arthrodesis with patellar graft using Hibbs', Galloway's, or Henderson's technique.
- (3) With a tibial graft using Britain's or Putti's technique.

The arthrodesis is usually performed with the knee in 5° to 10° of flexion, but if there is any doubt as to the stability, full extension is best. The main disadvantages are that there is a certain amount of shortening and a permanently stiff joint. It is contra-indicated in children under 8 years of age, as it interferes with epiphyseal growth.

Deformities of the Foot

As pointed out by Gill (1939), the object of all operations on deformed feet is to bring about a return to the normal appearance and a normal weight-bearing function.

Transplantations of tendons in deformities of the foot are

Severe cases are best treated by longitudinal traction with an additional pull from behind the tibial head.

Any other accompanying deformity such as flexion of the hip or genu valgum, should be corrected before a transplant is carried out.

Before transplanting any flexor tendons, one must be sure that the gastrocnemius or another flexor muscle is functioning. If operation is considered necessary, this usually entails a posterior capsulotomy and a lengthening of the hamstring tendons.

Dunn advised transplantation of the sartorius or tensor fasciae femoris to the patella, according to the local individual muscle balance, and Manfredi advocated transplantation of the sartorius, biceps femoris and tensor fasciae femoris.

Schwartzmann and Crego found that hamstring substitution for residual quadriceps paralysis was highly satisfactory, and that simultaneous transplantation of the biceps femoris and semitendinosus to the patella, produced a far better result than transplantation of the biceps femoris alone.

Transplantation of the ilio-tibial band and biceps is suitable for cases with quadriceps paralysis, combined with flexion of the hip and knee joints. The hip joint contraction is corrected four weeks after the transplant, and the knee joint contracture at a still later date.

Genu Recurvatum. This develops in cases with paralysis of the quadriceps, when the patient, fearing that the knee will give way, attempts to make the leg stable for weight bearing by locking the joint in hyperextension. The posterior part of the capsule and the posterior muscles are stretched and there is a contracture of the quadriceps tendon. It may also result from paralysis of the hamstring muscles.

If this condition is so severe that it is not controlled by a caliper or by Jones's knee brace, then operation is advised. Campbell implanted the patella into the tibia to produce a bony block and so prevent hyperextension. Supracondylar osteotomy with backward rotation of the lower fragment, or Irwin's tibial osteotomy have also been advocated. Irwin (1942) described this simple operative procedure in which the deformity is corrected without opening the knee joint.

The flexor hallucis longus and peroneus longus are inserted into the tuberosity of the os calcis

(2) In mild cases, the transplantation of the peroneus longus or tibialis anticus into the tendo achilles

(3) Gallie's operation.

(4) Putti's operation

Bony block at the anterior aspect of the talus prevents undue dorsiflexion of the foot. It is only carried out in patients who are too young for Dunn's operation. This operation restricts excessive mobility by impingement whilst retaining the desirable ankle movement.

(5) Whitman's Astragalectomy

This operation may be used instead of a tarsal arthrodesis. The subastragaloid joint is obliterated and the foot moves only by flexion and extension, lateral mobility being abolished. It is also indicated in talipes calcaneo-varus and talipes calcaneo-valgus.

Talipes Equinus. This results from paralysis of the extensors of the foot, but may be compensatory for a shortening of the leg. A slight shortening of the tendo-calcaneus may be an advantage, where there is shortening of the limb, or in cases with quadriceps weakness. When a patient with a weak quadriceps bears weight on the affected limb, the tension on the gastrocnemius muscle locks the knee, thus increasing its stability. In some cases, the tendo-calcaneus must be elongated, preferably by an open operation to permit a relaxation and recovery of the weakened dorsiflexor muscles of the ankle. They should be lengthened only enough to allow the foot to be placed at right angles, because if they are lengthened too much, a flail foot results and the downward thrust of the foot is lost in walking. If the dorsiflexor muscles of the ankle are completely and permanently paralysed, a tenodesis or arthrodesis in addition to, but preferably preceded by, a lengthening must be performed. In a tenodesis, the inactive peronei and tibialis anticus should be implanted into the tibia.

used mostly in conjunction with stabilizing operations on the bones. In carefully selected cases, transplants may be used alone for peroneal paralysis in which the tibialis anticus is attached to the cuboid, but the reverse procedure with the peroneal tendons for minor varus deformity is less satisfactory.

Inversion and eversion of the foot is treated by operative fixation in the subtaloid articulations, with transplantation of active muscles to positions of more useful function. Stability on weight bearing which is most important can, however, only be obtained by bony ankylosis. Stabilizing operations may cure the deformity but also greatly reduce the movement in the joints. Before extensive stabilizing operations are performed on the foot, the condition of the hip and knee joints must be considered.

Arthrodesis is particularly indicated in complete paralysis of the peronei and extensors of the foot, and in talipes calcaneo-valgus. It may take the form of a mid-tarsal arthrodesis, a posterior subastragaloid arthrodesis or a triple arthrodesis of the tarsus. Goldthwait performed arthrodesis of the ankle and subastragaloid joint, whilst Steindler fused the above joints together with the calcaneonavicular joint for a flail foot, associated with a good knee.

Before advising any operation, one must determine:

- (1) The cause of the deformity.
- (2) The degree of power present in the muscles.
- (3) The potentialities for recovery in the muscles.

Deformity may be postural or it may be due to:

- (a) Imbalanced muscular control of the foot increased by body weight.
- (b) Muscular contracture.
- (c) Gravity.
- (d) Habitual faulty posture.

Talipes Calcaneus. This is due to paresis or paralysis of the gastrocnemius muscle. It is treated by:

- (1) Mayer's physiological tendon transplantation.

The flexor hallucis longus and peroneus longus are inserted into the tuberosity of the os calcis

(2) In mild cases, the transplantation of the peroneus longus or tibialis anticus into the tendo achilles

(3) Gallie's operation.

(4) Putti's operation

Bony block at the anterior aspect of the talus prevents undue dorsiflexion of the foot. It is only carried out in patients who are too young for Dunn's operation. This operation restricts excessive mobility by impingement whilst retaining the desirable ankle movement

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In mild cases the peronei and tibialis posticus may be transplanted into the dorsum of the tarsus.

If there is paralysis of the tibialis anticus, the extensor hallucis longus is transplanted into the base of the first metatarsal. This operation may be combined with an astragalo-scaphoid arthrodesis.

Treatment is by.

- (1) Mayer's physiological tendon transplantation.
 - (a) Transplantation of the peroneus longus through the sheath of the tibialis anticus to the internal cuneiform.
 - (b) Transplantation of the peroneus brevis to the base of the fourth metatarsal.
- (2) As for (1) with the addition of arthrodesis of the calcaneo-scaphoid joint.
- (3) Campbell's extra-articular check operation.
- (4) Gill also described an operation to limit the plantar flexion of the foot by means of an ankle bone block.
- (5) Lambrinudi's arthrodesis.

This was designed primarily for cases with complete paralysis of the dorsiflexors of the foot and the peronei. The foot is fixed with the astragalus in full plantar flexion, so that when it is lifted from the ground the forefoot does not fall. There must, however, be some muscle control of the knee and preferably active calf muscles.

- (6) Dunn's Operation

This provides stability of the tarsus, mobility of the ankle, and bone and muscle balance of the paralytic foot. It preserves mobility of the ankle joint, and the tarsus is stabilized by arthrodesis of the calcaneo-astragaloid, calcaneo-cuboid, and astragalo-cuneiform joints.

Talipes Varus This is due to paralysis of the peroneal muscles.

Treatment is by.

- (1) Mayer's physiological tendon transplantation.
 - (a) Tibialis anticus through the sheath of the extensor digitorum longus and peroneus tertius to the base of the fifth metatarsal.
 - (b) Extensor hallucis longus through the sheath of the peroneus brevis to the fifth metatarsal.
- (2) Tibialis anticus is fixed subperiosteally into the cuboid, on the lateral aspect of the foot.
- (3) Extensor hallucis longus is transplanted into the base of the fifth metatarsal, or the tibialis anticus into the cuboid and the external hallucis longus into the first metatarsal.
- (4) Whitman's astragalectomy.

This operation is indicated for a flail foot with varus deformity.

Talipes Valgus. This is due to paralysis of the tibial muscles. It is treated by.

- (1) Mayer's physiological tendon transplantation.
 - (a) Extensor hallucis longus through the sheath of tibialis anticus to the inner border of the foot.
 - (b) Peroneus longus through the sheath of tibialis anticus to the inner border of the foot.
 - (c) Extensor digitorum longus and peroneus tertius subcutaneously to the inner border of the foot.
 - (d) Flexor digitorum longus through the sheath of the tibialis posticus and inserted into the scaphoid.
- (2) The peronei inserted subperiosteally into the navicular on the medial aspect of the foot.
- (3) As for (2), with arthrodesis of the talo-navicular joint.
- (4) Hoke's arthrodesis.

This stabilizing operation is not carried out before the age of twelve years, and produces minimal shortening of the leg. The indications are the same as for Whitman's operation, but it is especially useful in valgus deformity. The hip and knee joints must be in a position to co-operate. Ankle movement is retained and the subastragaloid, calcaneo-cuboid and astragalo-scaphoid joints are stabilized.

Talipes Calcaneo-Valgus. This deformity is corrected by transplanting the peroneal tendon into the os calcis.

Talipes Equino-Valgus. In this deformity, the peroneus longus is inserted into the dorsum of the medial cuneiform bone and this operation can be combined with an astragaloscaphoid arthrodesis.

Talipes Calcaneo-Cavus. The peroneus longus and flexor hallucis longus are attached to the tendo achilles.

Pes Cavus. Duchenne suggested that this condition was due to a paralysis of the interossei and lumbrical muscles. For slight extensor weakness, a metatarsal bar is all that is required. If there is slight flexion of the forefoot, dorsiflexion of the great toe at the metacarpophalangeal joint and flexion at the interphalangeal joint, operative treatment is necessary. In a still further advanced stage, Steindler's operation, Dunn's operation, or an osteotomy of the tarsus is performed. In the severest degrees, a talectomy is performed or amputation of the foot may be necessary. This is rarely desirable and should be undertaken only if there are gross trophic and ulcerative changes in a severely shortened limb.

Pes Planus. This may be treated by a talo-navicular arthrodesis.

Elmslie's Arthrodesis for Calcaneus Deformity of the Foot. This popular operation is especially useful when the calf muscles are also paralysed. The first stage is to correct the cavus deformity of the foot by stripping the soft tissue and performing a talo-navicular arthrodesis, after which the foot is immobilized in plaster of Paris in extreme dorsiflexion. In the second stage, the posterior part of the subtaloid joint is arthrodesed through a posterior approach. In addition, a strip of the tendo achilles is attached to the back of the tibia so that the foot is held at 20° in a position of equinus. The tibialis posterior, flexor digitorum longus and peroneus longus and brevis are implanted into the back of the calcaneum.

Deformities of the Toes

Claw toe is due to a weakness or paralysis of the flexors or intrinsic muscles of the foot. If it is associated with

pes cavus, this should be corrected first, because after correction the clawing of the toes may be minimal or absent. The treatment is by:

- (1) Insertion of the tendon of extensor digitorum longus into the head of the metatarsal.
- (2) Transplantation of extensor hallucis longus into the neck of the first metatarsal bone, for clawing of the great toe.
- (3) A similar operation to the above on each metatarsal, for clawing of the second, third, and fourth toes, or Hibbs's operation, where the tendons of the extensor communis are sutured to the external cuneiform. Higg's spike operation may also be used.

A flail great toe is treated by fusion of the metacarpophalangeal and interphalangeal joints.

Hallux valgus is treated by Keller's or Mayer's operation.

Flail Leg

This may be treated by arthrodesis of the hip, knee, and ankle joints, or by arthrodesis of the ankle and hip joints and the use of a Thomas splint with a knee joint, or by arthrodesis of the hip and fixation of the foot in equinus.

As stated above, a very careful assessment of all cases coming to operation is necessary and any recurrence of deformity or disability may be due to one or more of the following causes.

- (1) Residual muscle balance, deformities of the skeleton and instability of the ankle joint improperly assessed preoperatively.
- (2) Soft tissue contractures inadequately corrected before performing arthrodesis or tendon transplantation.
- (3) Using the wrong operative procedure.
- (4) Careless or improper tendon transplantation or implantation.
- (5) Deformity due to the action of unopposed muscles.
- (6) Deformity due to temporarily paralysed muscles.
- (7) Inadequate postoperative fixation.

- (8) Infection occurring during operation or post-operatively.
- (9) Aseptic bone necrosis.
- (10) Periostitis, osteomyelitis, or bony sequestrum.
- (11) Inequality in the growth of the limb, due to interference with the epiphysis.
- (12) Arthritis.

(E) OPERATIONS ON THE UPPER LIMB

Full mobility of all joints must be maintained. No operation is indicated unless some use could be made of the hand. Arthrodesis of the joint should not be carried out in a patient under fourteen years of age.

Winged Scapula

This is corrected by Whitman's operation where strips of fascia are passed from the scapula to the spinous processes of the fourth to the seventh thoracic vertebrae. Transplantation of the pectoralis major into the vertebral border of the scapula also gives good results.

Recurrent Dislocation of the Shoulder

Once subluxation or dislocation of the shoulder occurs, the strength of the muscles

stage of Nicola's operation is that the biceps tendon may not be strong enough to prevent redislocation, and in children, drilling through the epiphysis should be avoided.

Deltoid Paralysis

Adduction contracture of the shoulder may result after paralysis of the deltoid and is best treated by tenotomy of the pectoralis major and latissimus dorsi.

Ober's muscle transplant operation is used, if there is good power in the triceps and the biceps, whilst Mayer uses the trapezius muscle and a triangular segment of fascia lata. In the latter, the trapezius, serratus magnus, pectoralis major,

rhomboids, and levator scapulae must all be active, and it is contra-indicated if there is subluxation of the shoulder joint.

If the scapula is immobile, fixation in abduction is contra-indicated, and tendon transplantation is contra-indicated if the shoulder joint is subluxated.

Brockway (1939) tied the scapulae together with fascia lata in an attempt to improve the abduction power of the shoulder when there is paresis of the deltoid and rhomboid muscles. This is one of the few operations which should be performed early in cases of poliomyelitis.

In the majority of cases, arthrodesis is the operation of choice, except in early childhood when a tendon transplant is preferable because bony formation is not sufficiently established under ten years of age.

The methods most commonly employed are those of Watson Jones, Putti, and Brittain. The shoulder should be arthrodesed in 50° of abduction, 20° of forward flexion, and 20° of internal rotation.

Brittain's low extra-articular operation, with a tibial graft laid across the axilla between the humeral shaft and the lateral border of the scapula, gives better results in a young subject not under ten years of age, because of the scapular mobility.

after arthrodesis.

The following disadvantages should, however, be mentioned.

- (1) The cosmetic results are poor.
- (2) The movements are limited.
- (3) Scoliosis may result.
- (4) Fractures may occur owing to its exposed position and the decalcification secondary to the paralysis.
- (5) It is not advisable under fifteen years of age owing to the large amount of cartilage in the articulation and the possibility of interference with the growth of the arm, from trauma to the upper epiphysis.

- (8) Infection occurring during operation or post-operatively
- (9) Aseptic bone necrosis.
- (10) Periostitis, osteomyelitis, or bony sequestrum.
- (11) Inequality in the growth of the limb, due to interference with the epiphysis.
- (12) Arthritis.

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Recurrent Dislocation of the Shoulder

Once subluxation or dislocation of the shoulder occurs, there is no further improvement in the strength of the muscles.

The three operations used most commonly in this condition are those of Clairmont, Bankart, and Nicola. The disadvantage of Nicola's operation is that the biceps tendon may not be strong enough to prevent redislocation, and in children, drilling through the epiphysis should be avoided.

Deltoid Paralysis

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Supinator Brevis Paralysis

For paralysis of the supinator or contraction of the pronator muscles, Steindler transplanted the flexor carpi ulnaris tendon into the lower end of the radius on its dorsal aspect.

Paralysis of the supinator muscles may also be treated by transplantation of the pronator teres and flexor carpi radialis.

Supination contracture of the forearm may be treated by tenotomy or osteotomy.

Paralysis of the Wrist

In paralysis of the extensor muscles, transplantation of the flexor tendons is usually carried out and may be combined with fusion of the joint in moderate dorsiflexion and with the forearm mid-way between pronation and supination.

The three operations most commonly used are those of Steindler, Jones, and Stiles.

(1) Steindler's Operation

- (a) The flexor carpi radialis tendon is brought around the radial border and secured to the extensor pollicis longus.
- (b) The palmaris longus is brought around the radial border, and secured to the abductor pollicis longus and extensor pollicis brevis.
- (c) The flexor carpi ulnaris is brought around the ulnar border and secured to the four extensor tendons of the fingers.

(2) Jones's Operation.

- (a) The flexor carpi radialis is brought around the radial border and attached to the extensor pollicis longus and extensor indicis.
- (b) The pronator teres is brought around the radial border and attached to the extensor carpi radialis longus and brevis tendon.
- (c) The flexor carpi ulnaris is brought around the ulnar border and attached to the four extensor tendons of the fingers.

Arthrodesis is done only to allow further desirable activities without limiting movements already present, and it is contra-indicated if the hand is useless.

Bilateral arthrodesis of the shoulder joint is definitely contra-indicated.

Biceps Paralysis

One of the most recent operations described for this is that of Clark in which the lower part of the pectoralis major is transplanted into the paralysed biceps.

The operation apparently preferred in the United States of America for permanent paralysis of the biceps and brachialis is the proximal transplantation of the common flexor origin from the medial epicondyle, but it is essential that this group of muscles is strong and the hand useful.

Flexion contracture of the elbow may be treated by tenotomy of the biceps tendon.

Triceps Paralysis

Paralysis of the triceps is less important than biceps paralysis because gravity assists in extension.

A good triceps muscle is necessary for the thrusting and pushing movement of the forearm and hand, and also for locking the elbow in extension when crutches are used.

Ober has devised an operation to allow the brachioradialis to substitute for a weakened triceps.

Paralysis of the Elbow

If paralysis of the flexor muscles is incomplete, tendon transplantation may be used, but if the elbow is flail-like, arthrodesis at 90° in mid-rotation of the hand is indicated.

Steindler's operation for flail elbow should be used when there is paralysis of the biceps, brachialis, and brachioradialis, and when the flexors of the wrist and fingers are intact.

In bilateral cases, one elbow should be arthrodesed at 70° and the other at 110° of flexion.

pollicis longus in an attempt to restore opposition, or the first and second metacarpals may be united by a bone graft.

Steindler brings the palmaris longus and the flexor carpi radialis tendons around the radial side and attaches them to the extensor pollicis brevis, the abductor pollicis longus and brevis, and the extensor pollicis longus, in an attempt to restore abduction and extension of the thumb.

To restore the function of the intrinsic muscles of the hand, slips of the flexor digitorum profundus tendons are attached to the lateral borders of the proximal phalanges.

Stiffness in the metacarpophalangeal joints may be treated by one of three methods:

- (1) Manipulation under anaesthesia.
- (2) Removal of the heads of the metacarpals
- (3) Removal of the proximal ends of the proximal phalanges.

"Dangle Arm"

If the upper limb is completely paralysed, we get a completely flaccid extremity. This condition is known as "dangle arm" and it may be treated by.

- (1) Arthrodesis of the shoulder.
- (2) Flexorplasty of the elbow
- (3) Tendon transplantation of the wrist and thumb regions.
- (4) Arthrodesis of the wrist
- (5) A plastic operation on the joints of the thumb.

Whichever method is used for tendon transplantation in the upper limb, it should be followed by adequate physical therapy to develop and re-educate the transplanted muscle.

BIBLIOGRAPHY

- BROCKWAY, A. (1939) *J Bone Jt Surg*, 21, 451
 GALT, A. B. (1939) *Ann Surg*, 109, 252

(3) *Stiles's Operation.*

- (a) The palmaris longus is attached to the abductor pollicis longus and the extensor pollicis brevis tendons.
- (b) The flexor carpi radialis is attached to the extensors of the fingers and thumb
- (c) The pronator teres is attached to the extensor carpi radialis longus and brevis.

If a return of power to the extensors is to be expected, a paralytic flexion contracture of the wrist should be corrected by lengthening its flexor tendons.

Arthrodesis should be done when there is a paralysis of the extensor muscles of the forearm and a flexion contracture of the wrist.

For paralysis of the flexor muscles, Steindler advocates the following.

- (1) The extensor carpi radialis longus tendon is brought around the radial side and is secured to the flexor pollicis longus.
- (2) The extensor carpi ulnaris is brought around the ulnar side and secured to the flexor digitorum profundus

Arthrodesis to 35° of dorsiflexion with neutral deviation should be done only when tendon transplantation has failed, and *Brittain's operation* is preferable, as it does not interfere with pronation and supination.

If there is a flail wrist but with activity in the hand and fingers, then arthrodesis should be performed. An important point to note is that there is usually an improvement of muscle power following the arthrodesis.

Paralysis of the Small Muscles of the Hand

To replace the action of the paralysed opponens pollicis, Steindler attaches the split tendon of the flexor pollicis longus to the base of the proximal phalanx. Bunnell, on the other hand, uses the flexor carpi ulnaris, palmaris longus, or any other available long muscle.

The palmaris longus may be transplanted into the abductor

Cyanosis and discoloration of the skin with mild to severe oedema and hyperhidrosis are replaced by a flushed, dry, and warm skin, and the gradual disappearance of the oedema. The mobility of the parts is also improved, and deformity tends to be diminished.

Ogilvie (1933) stated that operations on the sympathetic system were indicated in the case of a cold, blue, usually oedematous limb in which chilblains, sores, or deep ulcers develop every winter, or in a limb with considerable and rapidly increasing shortening.

If the limb is completely paralysed, a lasting improvement in the circulation cannot be obtained by sympathectomy, but it may be useful in improving temporarily the local condition. This will enable the surgeon to carry out an orthopaedic operation which would be otherwise impracticable.

In recent years it was also found that after a lumbar sympathectomy there was an acceleration in the rate of growth in a partially paralysed limb, and an elevation in skin temperature in a child. Cold, blue, and moist feet and legs may show a temporary improvement after sympathectomy of the second and third lumbar ganglia, but a preliminary blocking of these ganglia with 1% procaine hydrochloride should be done, and a ganglionectomy not proceeded with unless a positive result is obtained. It is true to say that in the majority of properly selected cases, there is often a dramatic initial improvement in the circulation, which alas is not maintained, but it is equally true to say that the condition very rarely returns to a state as bad as it was before the operation.

Electrical treatment is sometimes prescribed for these cases, some surgeons favouring interrupted galvanism whilst others favour sinusoidal current. There is definite proof that this form of treatment is beneficial by its effect on the vasomotor control of the limb.

Harris and McDonald (1936) pointed out that under appropriate circumstances, lumbar sympathectomy would diminish the shortness due to the interference in the growth in the lower limbs paralysed by poliomyelitis. This beneficial result was attributed to the increased blood supply which

CHAPTER XVI

TREATMENT OF CIRCULATORY DISTURBANCES OF THE EXTREMITIES

THE peripheral circulation can be improved by the employment of general and local measures.

Rest in bed with adequate sleep is essential in the early part of the treatment as vasodilatation occurs during sleep. The feet are kept warm with bedsocks or well-protected hot-water bottles, and they should be washed daily and thoroughly dried. Methylated spirits are applied and allowed to dry, and then lanoline is rubbed in to keep the skin soft, supple, and free from scales.

Active and passive exercises are encouraged and heat may be applied in the form of dry heat or hot baths.

When the patient starts to walk, he must wear a fresh pair of socks daily, and his shoes should be of soft leather and loose fitting. Great care should be taken in cutting the toe nails and corns, and if a blister appears, it should be

forbidden.

Paravertebral sympathetic nerve block was used by Collins *et al.* (1947) for the relief of the oedema and the vasomotor and sudomotor disturbances of the extremities. In the case of the lower limb, 2% novocaine was injected into the lumbar ganglia, and in the case of the upper limb, the stellate ganglia. One side only at a time should be done, in order to be able to compare the two sides. After the first injection, signs of sympathetic paralysis appeared, usually within ten minutes, and improvement lasted for approximately three days, this period being longer with each subsequent injection.

Pain, muscle spasm, and muscle tenderness may be completely relieved by this method of treatment and it may be used in the acute and convalescent phases, even in children.

CHAPTER XVII

PSYCHOLOGICAL TREATMENT

THIS most important part of the treatment should begin as early as possible after the onset of the disease, in an attempt to counteract the mental strain which some patients must undergo when they realize that suddenly in the matter of a few hours they have been transformed from normal healthy persons into seemingly helpless cripples

If possible, the patient should be admitted to a ward especially reserved for this type of case, because the patients will then see the progress made by their fellows and this is a great help to their morale

The importance of living in hospital along with other patients similarly affected and who believe that one day they will return to some form of remunerative employment, must be emphasized.

The younger patients especially must be guided in the early stages so that they can adjust themselves to a prolonged convalescence

The excellent psychological effect of pool therapy must be remembered. Patients who lie completely immobile in bed, even for weeks, find that they can seemingly move their paralysed limbs in water, and are thus encouraged to make further efforts. Also the upright position and walking can be commenced much earlier if pool therapy is adopted.

As the period of treatment of poliomyelitis is necessarily prolonged over several months, if not years, the importance of schooling must not be forgotten, and all special centres should have full teaching facilities

Amongst the adult patients, there is a need for education and instruction to enable them to support themselves financially and to lead an independent existence when discharged from hospital

The patient is taught to accept his handicap and to acquire poise, self-assurance, and dexterity. He thus obtains

followed the operation, and which was assumed to induce an accelerated rate of growth.

BIBLIOGRAPHY

- COLLINS, V. J., *et al* (1947). *New Engl. J. Med.*, 236, 694
HARRIS, R. I., and McDONALD, J. L., (1936) *J. Bone Jt Surg.*, 18, 35.
OGILVIE, W. H (1933). *Proc. roy Soc. Med.*, 26, 429

CHAPTER XVIII

POLIOMYELITIS COMPLICATING PREGNANCY

As in the other acute infectious diseases, poliomyelitis and polioencephalitis may be associated with pregnancy.

The first recorded case of poliomyelitis in relation to pregnancy which I have been able to trace is that described by Baudry in 1891, where a woman of twenty-eight years with paralysis of the right arm and leg gave birth to her baby without any special difficulty. Further cases were described by Foulkrod (1923), Miller (1924), and Morrow and Luria (1939).

Aycock (1941) estimated that the probability of pregnancy and poliomyelitis coinciding was less than one in a thousand cases of poliomyelitis, and less than one in fifty thousand pregnancies. In the 1947 epidemic, 9.3% of women in the childbearing age group were affected, but there was not an unduly high incidence of the disease in pregnant women. Taylor and Simmons (1948) found that the incidence of poliomyelitis in pregnancy was twice as great in pregnant

between 20 and 35 years.

Jungeblut and Engle (1934) showed experimentally that hormonal changes during pregnancy make the animals particularly susceptible to infection by the virus of poliomyelitis.

Fox and Sennett (1945) reported that they had been able to trace eighty-five cases of pregnancy complicated by poliomyelitis, and they concluded that pregnancy increased the susceptibility to poliomyelitis due to endocrine upsets of the ovarian, pituitary, and foetal hormones.

Taylor and Simmons have suggested that this may be due to the increased congestion and permeability of the upper respiratory and digestive tracts which allow the virus an easier portal of entry during pregnancy.

■ proper valuation of his physical self and to a large extent is able to lose his self-consciousness.

Severely paralysed patients often become very depressed at the thought of leaving hospital, because they feel incapable of standing up to the stress and strain of everyday life. This difficulty can to a large extent be overcome by allowing patients to go to their homes on week-end leave, whenever they become ambulant and their condition permits. This helps them to become more self-reliant as there they can mix with their friends and relatives and gradually come to realize that they are not nearly so handicapped ■■ they had at first imagined.

have been so described, the evidence given to substantiate intra-uterine infection had been quite inadequate and inconclusive. In other words, they denied that intra-uterine infection could occur.

Biermann and Piszczek (1944) described a case in which the mother developed symptoms on the first day of the puerperium and died on the 4th day. The child had paralysis of both legs on the 13th day, but infection may have occurred at delivery from faecal contamination.

Wright and Owen (1952) reported on a pregnant woman who developed poliomyelitis nine days after her sister-in-law was admitted to an isolation hospital with poliomyelitis. Normal delivery followed as the legs only were involved. The patient died on the 6th day of the puerperium. The child weighed 7 lb at birth, but poliomyelitis developed on the 8th day and it died on the 10th day.

There is, however, definite evidence that the majority of new-born children have antibodies which protect them from infection in the first few weeks of life. This may explain why poliomyelitis in children under 6 months is rare.

Gillespie (1941) recorded a successful case of Caesarian section in a young mother of 18 years, but this operation is indicated only in very severe cases, particularly if respiratory paralysis is present, as it is usually the only means available to obtain a viable child when the death of the mother is imminent. Normally poliomyelitis in the mother does not interfere with the normal process of labour or spontaneous delivery, and the only indications for interference are for those complications which are liable to occur in any pregnant woman. Grelland (1947) states that of 35 patients who went into labour, 32 were delivered normally. The lack of voluntary muscular effort does not contra-indicate vaginal delivery and the degree of maternal spinal paralysis has no serious effect on labour. This is due to the involuntary contractions of the uterus which will expel its contents in experimental cases, even if the cord has been completely divided, and its sympathetic nerve supply removed.

Poliomyelitis victims who have become pregnant more than

Harmon and Hoyne (1943) observed that pregnancy had little or no effect on the extent of the paralysis in the mother. In one of their cases in which death of the foetus occurred from asphyxia due to extensive maternal paralysis, they were unable to isolate the virus from the spinal cord.

Weaver and Steiner (1944) noted that the incidence of poliomyelitis in pregnancy varies according to the stage of the pregnancy. In a small series of seventy cases, the incidence was as follows.

First third of pregnancy	17.1%
Second third	34.3%
Last third	48.6%

In Taylor and Simmons's cases, 76% occurred in the first six months of pregnancy. Of the 24% who developed poliomyelitis in the last three months of pregnancy three died, and the other three had severe residual paralysis.

Aycock (1946) feels that there is a tendency for the disease to occur in the first trimester of pregnancy if the foetus is a male, and in the third trimester if the foetus is a female, but judgment on this statement should be reserved.

Aycock and Ingall (1946) reported on six cases of abortion which occurred in the first three months of pregnancy.

It is now generally held that age, number of previous pregnancies and the stage of the pregnancy do not appear to be factors in the susceptibility of the pregnant woman to poliomyelitis. It is generally accepted that there is a higher incidence of abortions in women who contract poliomyelitis during pregnancy than in the normal pregnancy. This, however, is quite usual in several of the other infectious diseases. The incidence of bulbar involvement is definitely increased in the last trimester of pregnancy and may be as high as 23%. Bulbar poliomyelitis shows no tendency to result in abortion, premature labour or to cause precipitate labour.

In 1933 Brahdy and Lenarsky stated that in no instance in which a pregnant woman was affected by poliomyelitis was the disease transmitted to her baby, and in cases which

a year after their acute attack may be expected to have a normal pregnancy and labour and the baby to be normal.

Statistics tend to show that no advantage is to be gained in interrupting pregnancy at any stage in these cases unless the poliomyelitis has occurred several years previously, causing pelvic deformity due to unilateral dislocation of the hip or paralysis of one leg. Pregnancy may, however, increase the severity of the poliomyelitis or its complications such as cystitis or diaphragmatic paralysis.

In our series of 500 cases, 17 were found to be pregnant. On close examination of their signs and symptoms, there does not appear to be any marked difference between the pregnant and the non-pregnant patients, e.g. 53% were feverish, 35% had headache, 41% had loss of appetite, whilst 59%, 53% and 41% had pain in the back, pain in the limbs and pain in the neck respectively.

The time between the onset of the illness and the first appearance of the paralysis varied from one to ten days and it was sudden in onset in 66% of cases.

The distribution of the paralysis was widespread, but in only two cases were the cranial nerves involved. These included the right facial and glossopharyngeal.

The duration of pregnancy at the onset of poliomyelitis was as follows:

at 3 months	4 patients
at 5 months	1 patient
at 6 months	4 patients
at 7 months	2 patients
at 8 months	3 patients
at 9 months	3 patients

The termination of pregnancy did not appear to depend upon the extent of the paralysis, but upon the effect of the disease upon the general condition of the patient. It would therefore appear that the effect of poliomyelitis on pregnancy does not differ a great deal from any other acute infective lesion.

BIBLIOGRAPHY

1944-1945 *Brit. med J* 2, 405

Sci, 212, 366.

med Ass, 101,

BIERMANN, A. H., and PISZCZEK, E. A. (1944) *J. Amer med Ass*,
124, 296

FOULKROD, C (1923) *Amer J Obstet. Gynec*, 5, 327

FOULKROD, C (1945) *Amer J med Sci*, 209, 382

Center, 3, 22

Ass, 123, 185

Med, 59, 43

med Ass, 113,

TAYLOR and SIMMONS (1948). *Amer J Obstet Gynec*, 56, 143

WEAVER, H. M., and STEINER, G (1944) *Amer J Obstet Gynec*,
47, 493.

WRIGHT, G. A., and OWEN, T. K. (1952) *Brit. med J*, 1, 800

CHAPTER XIX

DISCHARGE

BEFORE discharge, the muscles should be manually tested, and a record made of the ability of the patient to stand, walk or climb stairs, and any abnormalities in the gait should be noted.

A printed home treatment programme should be given to each patient or to his parents, and the physiotherapist should explain this in detail so that the patient knows exactly what exercises he has to carry out before next attending as an out-patient. It is essential that as far as possible the same physiotherapist should attend at each subsequent clinic so that the patient's progress may be properly assessed.

In the case of children, the parents should be made fully aware of the child's capabilities and limitations, so that he can enjoy supervised physical recreation as well as intellectual training. Games in which balance is controlled mainly by the unaffected muscles should be encouraged, e.g. diving, bicycling, and horse riding for patients with a lower limb paralysis. Golf is an excellent exercise for the older children where the shoulder muscles are weak. Swimming can also be taught to children even if there is paralysis of the legs, because in the crawl, for example, most of the propulsive power comes from the arms. The aim in all these activities should be that of achievement because failure in competitive sports may completely dishearten the patient.

On the aesthetic side, long trousers may hide the paralysed limb and give the young wearer a feeling of superiority.

Ordinary schools are preferable for the physically defective as the emphasis must be placed upon the capacity of the patient and not upon his disability.

Patients between the ages of 16 and 21 years should be trained in occupations in which they will be able to earn a livelihood, and compete on a more or less equal footing with able-bodied workers.

Under the Disabled Persons (Employment) Act of 1944,

the Government are under an obligation to find suitable employment for all disabled persons, including those paralysed by poliomyelitis

Even the mildest cases should be reviewed every month for three months and the interval then gradually extended to three months. A follow-up extending over at least five years is most desirable because late deformities such as scoliosis, talipes equinus, pes cavus, etc., can then be detected in the early stages

At each attendance in addition to the general clinical examination, the patient should be questioned as to his progress, abilities and disabilities, his posture and gait are noted, and splints and appliances checked. His chest expansion and vital capacity are recorded and also the condition of the abdominal muscles.

During the first year, slight defects in position which impede restoration of function can be corrected by daily stretching, slow steady tension or by gradual stretching by suitable splintage. Forceful manipulation under anaesthesia is most undesirable and dangerous, because loosening, subluxation or dislocation of the epiphysis may result. If forceful manipulation is used, there is a marked reaction with pain and swelling and severe limitation of movement. This should be treated by rest until the reaction subsides.

CHAPTER XX

PROGNOSIS

As far back as 1896, Courtney published a paper in which he drew attention to the fact that the prognosis depended upon early diagnosis and treatment. He also showed that a patient with a very extensive primary paralysis may have less residual paralysis than one who had a less extensive primary paralysis, and that in his opinion, improvement could be expected for anything up to four years after the attack.

Broadly speaking, it might be said that the prognosis depends upon

- (1) The resistance of the patient.
- (2) The virulence and type of the virus
- (3) The portal of entry
- (4) The area involved in the central nervous system.
- (5) The amount of destruction in the central nervous system.
- (6) The rate of development of symptoms.
- (7) The duration and degree of the fever.
- (8) The amount of physical activity during the various stages of the disease.
- (9) The lapse of time between the initial invasion and the commencement and duration of specific treatment.
- (10) The onset of other complications.

In the abortive type of case, the prognosis is excellent and the patient invariably makes a complete recovery from all his symptoms in four to seven days. In the preparalytic stage, however, it is impossible to foretell if the patient will develop paralysis, or if he does, to what extent, and what will be his ultimate disability.

Even in the early convalescent stage, accurate forecasts about recovery are impossible as a large percentage of cases recover completely or almost completely even without any treatment.

The motor nerve cells which have not been completely destroyed are thought to begin to resume their function in ten to fifty days after the initial attack. The paralysis will therefore diminish in extent, but if the cells have been permanently destroyed the related muscles will begin to atrophy. It is therefore obvious that the completeness of the recovery depends largely upon the original damage to the nerve cells, and the extent of this recovery can be ascertained only by repeated examinations extending over a period of up to two years.

It has been estimated that from 50% to 80% of all cases make a complete recovery, 15% to 45% are permanently paralysed to a varying degree and that 5% to 10% die. The great diversity in these figures is undoubtedly due to the number of abortive and preparalytic cases which are missed clinically or have been included by other observers in their final figures. In our series, the comparative figures were as follows:

Complete recovery	63%
Partial recovery	24%
Minimum recovery	10%

In the New York epidemic of 1931, 82% of all cases admitted to hospital in the preparalytic stage did not develop paralysis.

It should be pointed out that it is useless to compare the improvement in different patients simply by calculating the improvement between the patient's first visit and subsequent visits, without knowing first of all the interval of time between the onset of the paralysis and the date of the first visit to hospital. This is indeed true, as the maximum improvement occurs during the first six months, and if the patient does not attend for some weeks or months after the initial attack, an entirely wrong impression may be formed of the amount of recovery which had already taken place and that which may ultimately result. It can therefore be reasonably concluded that a patient who has a certain degree of paralysis one month after the onset has a better prognosis than a

patient who has a similar degree of paralysis three months after the onset.

In order to determine the relative improvement in any two groups of patients, it is necessary to compare only the same degree of original paralysis. If therefore we take two groups of patients and examine the first group at the onset of the disease and again one month later, and the second group at the end of the first month and again one month later, we would expect, if the two groups had a specific amount of paralysis on the first examination, that the first group would have much the better prognosis. If this is done, it is found that the prognosis is much better if the patient comes under early adequate supervision.

Johnson (1945) stated that the clinician should be able to give an accurate prognosis at the end of the first three months. He estimated the recovery of the muscles in his cases in percentages and expressed his results at the end of that time as follows:

- (1) Muscles which have not recovered 30% of their function will have no useful power.
- (2) Muscles which have a return of between 30%-75% of their function require expert treatment for at least one year.
- (3) Muscles with a return of 80% of their power will function satisfactorily and will require no special treatment beyond general supervision.

He also stated that regression can occur from over-fatigue and too early removal of supports, and he concluded that no appreciable recovery continues after eighteen months.

In the classification used in this country, it is the opinion of some observers that after three months of adequate supervised treatment, those muscles in group (2) or in a lower category are most unlikely to develop any useful power, and that the only treatment now is that required to prevent any subsequent deformity. In our opinion, this observation is quite unwarranted especially if one considers functional recovery and muscle balance.

The muscles in group (3) may be expected to improve further and should ultimately have a useful function. Their power may be increased by appropriate treatment to develop hypertrophy of the remaining active muscle fibres and by paying particular attention to the avoidance of fatigue and postural strain.

Muscles in group (4) should eventually function satisfactorily, if adequate supervision is continued.

Complete mental and physical rest in bed during the abortive, preparalytic or early paralytic stages of the disease may prevent or greatly ameliorate the resultant paralysis, but prolonged immobilization is harmful because of the danger of disuse atrophy and subsequent joint stiffness.

If the superficial and deep reflexes are not lost, and there is only minimal weakness, then the patient is very likely to make a complete recovery. In fact, in these cases, improvement in the muscle power may be noticed within a few hours from the onset.

The early return of voluntary muscle power or of the reflexes are hopeful signs, and those muscles which show a good initial improvement will usually become strong with appropriate treatment. According to Schwab *et al* (1947), it is four to six months before any substantial increase in the size and strength of the paralysed muscle can be clearly demonstrated.

Useful recovery within the first month after the onset of the disease can be seen, but we may get progressive improvement over a period of two to three years. Early recovery is due to the reversal of the transient changes in the spinal cord and brain stem. Late recovery appears to be due to a compensatory hypertrophy of the normal muscle fibres or to a compensatory branching of the intact motor nerve fibres.

The rapidity with which the symptoms develop and progress also seems to be of importance, because a prolonged first stage is often followed by a markedly delayed or incomplete recovery. Also if the muscle tenderness lasts for some weeks, improvement is delayed until it disappears. Advance of the paralysis is to be feared as long as fever is present, but a progressively falling temperature generally indicates

that the paralysis is now maximal. Extensive and severe paralysis may, however, develop unexpectedly and rapidly, even in the absence of pyrexia. Whilst fever persists prognosis in respect of paresis must be guarded.

If the attack is extremely virulent, the paralysis may increase over several days, that is if the patient lives long enough for it to be observed. An initial paralysis of all four limbs does not necessarily imply that a complete recovery is impossible. Paraplegia may occur, but if only one limb is affected, the left is more frequently involved than the right. The arm is usually less severely involved than the leg, and it usually recovers more rapidly and completely. The right hand is also said to recover to a greater degree than the left. If a complete paralysis of one or more limbs or a whole segment of a limb persists for three to four months, then the prognosis is bad.

Unfavourable signs to watch for are marked and persistent pyrexia, drowsiness and severe prostration, marked cerebral irritability and extensive paralysis, especially if this involves the respiratory muscles, the accessory muscles of respiration and the respiratory centre.

In our cases, the approximate time between the onset of the illness and the time when the paralysis first showed signs of recovery was as follows:

1st week	47%
2nd week	27%
3rd week	24%

Generally speaking, the higher the lesion in the spinal cord, the worse the prognosis as regards life, but the better for the recovery from the peripheral paralysis.

The paralysis of the respiratory muscles may be only temporary, and if these cases are treated in a mechanical respirator during the period of paralysis, the muscles may partly recover and so prevent an immediate fatal issue. If bronchitis or pneumonia develop, or mucus or vomitus is aspirated into the lungs, the outcome is almost invariably fatal. Should there be no real degree of recovery of the severely paralysed intercostal muscles within the first three

months, the great majority of these patients will die within two years from an intercurrent lung infection.

Directly and indirectly, the principal cause of death in the older age groups appears to be the respiratory involvement.

Cases which show primary circulatory failure without any evidence of gross respiratory involvement are invariably fatal.

Paralysis of the bladder is frequently described in the literature, but recovery is invariably complete.

Age does not appear to have any influence on the rate or degree of recovery although certain observers have stated that the maximum recovery takes place between the ages of 15 and 20 years.

Deformities and contractures may occur later as the result of gravity, the unopposed pull of the normal muscles, and the contraction of the fibrous tissue in the paralysed muscles. It is impossible to make an accurate diagnosis or to give a reasonable prognosis until deformities have been corrected, because these contractures interfere seriously with the return of muscle power and function. Also if the joint is unstable, the muscle cannot act to its maximum capacity, and therefore an attempt should be made to stabilize the joint before trying to determine accurately the remaining power of the muscle. It has also been shown that any patient can be made to walk if his deformities are corrected and if he can support himself in the erect position on crutches.

Many patients who survive with extensive paralysis and muscular atrophy suffer greatly from cyanosis and coldness of the limbs which may later show evidence of chilblains and trophic lesions.

In cases with bulbar involvement, the prognosis is particularly good in that group where there is involvement only of the cranial nerve nuclei. Death is due to the involvement of the cardiac and respiratory centres in the medulla and occurs in approximately 50% of such cases.

If nystagmus alone is present, the prognosis is excellent, because if the patient lives, this clinical finding will disappear within three months. The prognosis for facial nerve paralysis is moreover not nearly so good.

From the above it will be seen that at present there are no

efficient methods for the control of an epidemic, the prevention of paralysis, or the cure of the disease. All that we can do is to try to repair the damage.

BIBLIOGRAPHY

- COURTNEY, J N (1896) *Boston med surg. J.*, 135, 617.
JOHNSON, R W, JNR (1945) *J Bone Jt Surg*, 27, 223
SCHWAR, R S, *et al* (1947) *Trans. Amer. neurol. Ass.*, 72, 137.

CHAPTER XXI

MORTALITY AND MORBIDITY

FINALLY let us consider briefly the question of mortality and morbidity

Dornedden (1933) gave the following mortality rates for epidemics which occurred in Germany during the years 1927-32.

Year	No. of cases	Mortality %
1927	2,840	12.8
1928	996	15.1
1929	1,157	15.0
1930	1,363	9.5
1931	1,613	11.2
1932	3,735	8.4

It will be seen that the above table does not take into consideration the age of the patient which is a most important point when discussing the mortality rate in poliomyelitis

Craster (1916) reported on a series of 1,360 cases in which there were 363 deaths. In this series 80% of the deaths were in children under five years of age.

Blencke (1933) reported on 1,695 cases with a mortality in the age group 1-5 of 4.02% and in the older age groups of 8.94%

Seddon (1943) reported on the Malta epidemic involving 460 persons, giving a mortality rate of 3.5% in children and 17% in adults

In the British epidemic of 1947, there were 333 deaths giving a mortality rate of 8%, of which 14% occurred in the 0-5 age groups, 21% in the 5-15 age group and 65% in the over 15 age group. Of the total epidemic mortality, 70% occurred in adults.

Dauer (1948) also found that the mortality rate had markedly decreased in the age group under 5 years, but that it had increased in the other age groups.

In the outbreak in Canada amongst the Eskimos in the severe winter of 1948-9, there were 60 cases with 18 deaths, giving a mortality rate of 30%. It should be noted that there were no deaths in children under 8 years of age whilst 5 of the patients were over 60 years of age.

In the Swedish epidemic, it was found that the case mortality rate was higher in the towns than in the country districts.

Death is commonest on the fourth day and 90% occur during the first fortnight of the illness usually from acute respiratory failure.

In our series, 13 cases died. Of these, 2 were under the age of 5 years, 3 were between 5 and 15 years and 8 were over 15 years of age. Of the 13 deaths, 9 were males and 4 were females. Death occurred in 9 cases within the first five days of the illness usually from respiratory failure.

During the 1947 epidemic, there were 3,461 paralytic cases and 3,128 cases who recovered; 41.1% were classed as mild, 38.5% as moderate and 20.4% as severe. Of the 1,483 survivors in the moderate and severe classes, the age grouping was as follows:

Age	No. of cases	Percentage
0-1 years	102	5.5
1-5 "	586	31.8
5-15 "	588	31.9
15-25 "	295	16.0
25-35 "	192	10.3
35-45 "	68	3.7
45+	12	0.7

Forty per cent of the above cases will require prolonged treatment in hospital. There is a preponderance of males over females of about 5 to 4; 310 patients were treated in respirators and 93 may require such treatment permanently.

BIBLIOGRAPHY

- BLENCKE, A (1933) *Z Kruppelfürs*, 26, 169.
CRASTER, C V (1916) *Trans Amer Ass Study Prev Inf Mortal*,
7, 187.
DAUER, C C. (1948) *Amer J. Hyg*, 48, 133.
DORNEDDEN, H (1933) *Arch soz Hyg*, 8, 187.
SEDDON, H J. (1943) *Lancet*, 2, 549.

CHAPTER XXII

COMPLICATIONS

POLIOMYELITIS may be complicated by a large number of factors which affect particular systems in the body, but only a brief outline of the commoner and more important ones will be given.

1. *Fractures.* Berbez (1888) described a case of spontaneous fracture in a limb affected by poliomyelitis, and Davis and Yoffa (1927) reported on another case in which a fracture of the femur occurred during the course of treatment in a child of two years of age.

Normally, these fractures are due to trauma which may be of a trivial nature only. In long-standing cases with paralysis, the bones are atrophied and the callus is not strong because of the functional disuse and the absence of good muscle tone around the bone. These points were discussed fully by Tumpeer and McNealy (1930).

2. *Dislocations and Subluxations.* The first case of a dislocation complicating poliomyelitis was recorded by Ridlon in 1898 in which the hip was involved. The two most common sites for dislocation and subluxation are the hip and shoulder joints, and this complication should always be looked for, when the patient complains of pain in these areas

3. *Bony Deformities.* These may be assumed to arise from one or more of the following causes:

- (1) Disturbances in growth in children which later give rise to deformities.
- (2) Over-fatigue and stretch paralysis.
- (3) General exercises without regard to muscle balance.
- (4) The effects of gravity.
- (5) Habitual incorrect posture.

- (6) Unprotected premature functional use.
- (7) Weight bearing without mechanical support in positions favouring deformity.
- (8) Muscle imbalance as shown by a combination of muscle spasm and weakness in the earlier stages.
- (9) Contractures due to fibrous contracture in the later stages

According to Barr (1948), approximately one-third of all patients with poliomyelitis which occurs before the age of sixteen, develop marked inequality in the length of the lower limbs. The determining factors in these cases appear to be the age of onset, the sex, and the amount and distribution of the muscular weakness of the legs. It should be noted, however, that there is no shortening if the distribution of the paralysis is symmetrical, whilst the greatest shortening occurs in young patients with one normal and one severely paralysed leg. Premature cessation of growth associated with epiphyseal closure on the paralysed side, may explain the late occurrence of marked differences of length. The possible causes of the unequal growth may be the retarded circulation, trophic disturbances and disuse of the extremity, but it is still impossible to predict the actual amount of shortening. This shortening may be corrected by

A Elevation of the sole of the shoe, if the short-

B

it,

- (1) There is a shortening of over 2.5 cms. in which no compensation is possible by pelvic tilting.
- (2) There is a shortening of over 4-5 cms. where there is a mobile pelvis.

Contra-indications are:

- (1) Age. Only adolescents and young adults are suitable.
- (2) Poor circulation in the affected limb.
- (3) Active bone or joint disease.

- (4) Marked shortening where amputation would give a better result.

The commonest dangers and complications are:

- (1) Infection of the wound which may lead to osteomyelitis.
- (2) Damage to the nerves and vessels.
- (3) Mal-union, delayed union, or non-union of the bone
- (4) Increased weakness of the lengthened muscles.
- (5) Fracture.
- (6) Oedema of the limb.
- (7) Stiffness of the joints.

- C. Diminishing the length of the femur and/or the tibia and fibula of the normal leg
- D. Epiphyseal arrest in children and adolescents by epiphyseodesis, but the operation should be performed preferably before the age of 12 years.
- E. Lumbar sympathetic ganglionectomy. The maximum result in this operation is obtained if it is performed before the age of 6 years, but the total improvement in length is only about one inch (Harris, 1930).

Neurath (1900) described a case of poliomyelitis in which the bones of the paralysed extremity were elongated.

Andérodias (1900) and Schmoucler (1913-14) studied the structural pelvic deformities due to poliomyelitis, and the latter discussed their obstetrical significance which is now well recognized. Asymmetry of the pelvic inlet is said to be present in 80% of patients who suffer from paralysis of the lower limb due to poliomyelitis. Pelvic obliquity which is due to contractures can also cause faulty posture

4. *Arthritis.* Poynton (1943) stated that in his opinion,

and wrists, but there is no local evidence of redness or heat. Nevertheless he states that some of the joints affected may undergo fibrous ankylosis after several months.

In chronic cases, a radiographic examination shows thickening of the capsule, diminution of the joint spaces, and some rarefaction of the bone. Suppuration never occurs, and as a rule, complete recovery will take place with conservative treatment.

5. Contractures The importance of the early prevention of contractures and stiff joints cannot be stressed too often or too forcibly. On admission to an orthopaedic hospital, poliomyelitis cases are sometimes found to have early contractures and limitation of movement of the joints. On enquiry it is invariably noted that such cases have had no splinting or physiotherapy treatment in the early stages. It is a well-known fact that if muscle weakness is not adequately controlled, it will cause faulty alignment, resulting in contractures and deformities. In consequence, much valuable time is lost in correcting deformities which should never have been allowed to occur.

The contractures which appear most frequently are those in the hip, knee, foot, shoulder, elbow and hand, and they are particularly to be looked for if the extensors of a joint are paralysed and the flexors are normal.

Certain observers attribute the deformity to overaction of the unaffected muscles in opposition to the weakened or paralysed ones.

Physiological contractures may be associated with:

- (1) Muscle spasm and temporarily denervated muscles
- (2) Functional changes at the neuromuscular junctions associated with a hypersensitivity to acetylcholine
- (3) Changes in the central nervous system associated with muscle spasm.

Pathological contractures which result from muscle atrophy and fibrosis are brought about by peripheral inflammatory reactions taking place in the tissues, particularly in the muscles. The terminal results of these changes are muscle atrophy, fibrosis and persistent irreversible contraction of the muscle. These pathological changes are insidious in onset and frequently give rise to the development

of excessive fibrosis which causes shortening of the muscle as a whole and a limitation of its power of extensibility and relaxation.

A muscle may have sufficient power to function satisfactorily if there is no deformity, but if contractures and stiff joints are present, its function is further inhibited. In cases with joint stiffness, the muscle develops an adaptive shortening, but if there is complete loss of joint movement, the muscles will undergo disuse atrophy. In growing children, the deformity tends to progress, because of the effects of the growing bone.

If there is any tendency to contractures, they should be treated immediately by gentle but firm stretching and by adequate splinting, so that the weakened muscles are supported in their normal position. If splints or appliances are already being worn, they may be found to be inadequate or ill-fitting and immediate adjustment is essential. Established contractures can usually be corrected by braces, a series of plaster casts, gentle and prolonged traction or by operation.

The two contractures most commonly missed are those of the ilio-tibial band and of the rectus femoris. Contraction of the former may result in.

- (1) Flexion and abduction deformity of the hip.
- (2) Pelvic obliquity and a secondary spinal curvature when the patient assumes the erect posture.
- (3) Flexion deformity of the knee.
- (4) Genu valgum
- (5) External rotation of the tibia with or without a subluxation on the femur.
- (6) Contraction of the intermuscular septum producing disturbed growth on the lateral aspect of the knee

Contraction of the rectus femoris may cause flexion contractures of the hip, lordosis and scoliosis. Knee flexion is also limited, but it may be masked by flexion of the hip.

It is interesting to note that progressive muscular atrophy may occur as a rare sequel to poliomyelitis which it may

follow after several years. In these cases, the progressive muscular wasting usually begins in the region originally affected.

6 Tuberculosis Valentin (1935) commented upon the apparent immunity of poliomyelitis patients to tuberculosis, and Rumshina (1933) reported upon the second case in the literature of the combination of joint tuberculosis and poliomyelitis

7 Circulatory System Local neuromuscular changes such as coldness, blueness, swelling and congestion occur in cases with severe paralysis, and they can cause considerable disability. Lumbar ganglionectomy may give relief for a few years, and during that time the surgeon can correct deformities and other abnormalities unhampered by the presence of sores and ulcers.

Abramson *et al* (1943) determined the blood flow in the paralysed extremities but failed to discover any marked change between the circulation in the affected and normal limbs, and peculiarly enough found that in a few cases the circulation could be actually greater in the paralysed limb. They also observed that the cutaneous blood-vessels of the paralysed limb responded much more rapidly to changes of temperature.

Phlebitis is another troublesome complication which should be kept in mind.

Gross oedema is rarely present, but local oedema has been noticed in various parts of the body including the lower limb and shoulder girdle. Mussio-Fournier (1928) reported on a case in which there was an intense symmetrical and transitory oedema at the level of the calf. Brehme (1932) described a temporary oedema of the left instep in a child 6 months old, three days after the onset, and followed almost immediately by paralysis of the left arm and leg. The oedema became more marked on the left foot, over the left tibia and on the dorsum of the left hand, but it regressed slowly as the acute phase passed. He assumed that these changes were due to an alteration in the spinal trophic centres which

of excessive fibrosis which causes shortening of the muscle as a whole and a limitation of its power of extensibility and relaxation.

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it is usually seen in adults during treatment in a mechanical respirator. It is attributed to:

- (1) A general atony of the gastro-intestinal tract.
- (2) Lack of effective abdominal musculature
- (3) Aerophagy.
- (4) Over-feeding

It is treated by continuous gastric suction, intravenous infusions, and the injection of prostigmine 0.5 mgm t.d.s. or carbachol 0.5 cc. intramuscularly.

Baumel (1910) observed a case of megacolon in an infant suffering from poliomyelitis.

Heyde and Robinson (1948) described two cases of bulbar poliomyelitis in which one was complicated by multiple duodenal ulcers, and the other by a large perforation in the fundus of the stomach.

10 Renal System : Keyes (1937) and Wappler (1937) both described renal complications in cases of poliomyelitis. In the former's case, toxic nephrosis and pyelitis were present, whilst in the latter's three cases there were renal calculi. The main factors given by Wappler to explain the formation of these calculi were.

- (1) Retention of urine due to the poliomyelitis.
- (2) Paralysis of the abdominal musculature.
- (3) Urinary infection
- (4) Diminished peristalsis in the urinary tract with consequent stasis of urine
- (5) Diminished renal secretion
- (6) Disturbances in metabolism
- (7) Stasis due to decubitus

This last point was also stressed by Brady and Wilson (1948) who maintain that with immobilization of the patient, there is deficient drainage of some or all of the renal calyces.

This complication may be prevented by repeated turning in the respirator and if possible by frequent short periods in

regulate the lymph flow, thus leading to stasis of the lymph. Other observers have attributed its presence to the impaired lymphatic circulation in a grossly paralysed limb, and consider it to be secondary to the muscular inactivity.

In one of our cases, the leg became stiff and swollen two days after the onset of the paralysis, and in another where there was paralysis of the right deltoid, a swelling was noticed around the right elbow.

Oedema is a recognized cause of stiffness, and the swollen limb should be treated by elevation and frequent passive movements until the oedema has disappeared. Massage may also be helpful, and later, an elastic bandage or stocking should be worn.

Discoloration of the skin and chilblains are often accompanied by marked oedema. They are treated by exercises to the part for half an hour, followed by the application of paraffin wax to the affected part. Interrupted galvanism and sinusoidal baths are also helpful.

8 *Pulmonary System* . Lobar, hypostatic, aspiration and broncho-pneumonia have all been found as complications of poliomyelitis, and atelectasis may also occur from the blocking of a bronchus by inspissated sputum, secretions or vomitus.

9 *Alimentary System* . Constipation is a very common finding, and could be due to the abnormal position in which the patient must defaecate, or to the lack of activity. Paralysis of the abdominal musculature, if present, could be another contributing factor.

Diarrhoea may also occur and it is thought to be due to an attempt by the body to get rid of the irritating infected faeces.

Paralytic ileus has been observed and usually appears three days after the first signs of the paralysis of the voluntary muscles. There is very little or no pain, but the more severe cases are liable to be complicated by cardiac and respiratory embarrassment.

Acute dilatation of the stomach occurs infrequently and

high pressure in the cerebrospinal fluid which was present over a period of five months, and he advocated repeated lumbar puncture for the relief of the headache. In this particular case, bilateral choked discs were observed at the fourth week.

Gordon *et al* (1939) studied the effect of poliomyelitis on the intellectual capacity of the patient, and came to the conclusion that the level of general intelligence was normal. They did, however, point out that prolonged absence from school or maladjustments of personality may simulate true mental retardation.

Seven of our cases were complicated by the onset of either scarlet fever, chicken pox or measles three to five weeks following admission.

Lastly we may mention that a case of dystrophia adiposogenitalis has been known to occur after an attack of poliomyelitis.

BIBLIOGRAPHY

- ABRAMSON, H., et al (1943) *Arch intern Med*, 71, 391
ANDERODIAS, J (1900) *Gaz hebdom Sci med Bordeaux*, 21, 483
AYER, J B, and TREVETT, L D (1934) *Arch Neurol Psychiat*
(Chicago), 31, 396
BARR, J S (1948) *New Engl J Med*, 238, 737
BAUMEL, L (1910) *Rev gen Clin*, 24, 401
BERBEZ, P (1888) *Bull Soc clin Paris*, 11, 381
BRADY, L, and WILSON, W J (1948) *J Urol (Baltimore)*, 61, 381
BREHME, TH (1932) *Kinderärztl Prax*, 3, 553
BROWN, C and FLEMING, I (1933) *N Y Clin pediat*, 25, 780
Surg, 2, 166

GUTHRIE, J (1933) *Brit med J*, 2, 803
HALLION, L (1889) *France med*, 1, 318
HARRIS, H I (1930) *J Bone Jt Surg*, 12, 859
HEYDE, E C, and ROBINSON, S (1948) *Gastroenterology*, 11, 519
KEYES, H L (1937) *J Paediat*, 10, 233
MUSSIO-FOURNIER, J I (1928) *Bull Mem Soc med Hôp Paris*, 52,
424
NEURATH, R. (1900). *Wien klin Wschr*, 13, 563
PERPIGNANO, O G (1940) *G ital Derm sci.*, 81, 1003.
POYNTON, F. (1943) *Lancet*, 245, 433

the sitting or standing positions. If this is contra-indicated owing to weakness of the trunk muscles and possible scoliosis, then bed exercises and frequent changes are essential. The patient should also receive abundant fluids.

One of our cases was complicated by an attack of glomerular nephritis.

11. *Dermatological Changes.* The first description of skin changes was that by Hallion in 1889 when he reported on a case in which purpura appeared in the atrophied limb. Fox (1923) mentioned the atrophic changes which occurred in the skin after poliomyelitis, whilst Perpignano (1940) described a case where Mibelli's angiokeratoma appeared on the scrotum during the early period of paralysis. Two of our cases developed a petechial rash on the neck and body two days after the onset of the illness. An urticarial rash also appeared on the arms although no paralysis occurred there.

12. *Central Nervous System.* In 1882, Friedlaender described calcification of the ganglion cells in the anterior horns in poliomyelitis, but I have been unable to trace any further literature on this finding.

Redeschi (1903-4) reported on a case of poliomyelitis complicated by facial hemi-atrophy and atrophy of the optic nerve, and Gordon (1904) described another case with peripheral facial palsy and paradoxical pupils associated with hippus. Ehlers (1936) recorded a case in which hippus appeared simultaneously with Horner's syndrome, the latter condition being explained by a lesion of the ciliospinal centre and in the nerve fibres leading from it.

Tilney (1918) described a case of poliomyelitis in which the patient had prolonged somnolence, but the majority of patients suffer from insomnia and some cases from restlessness, hallucinations and confusion. One of our cases was semi-conscious for one week and another was delirious for four nights.

Busacca and Tomassini (1927) described a case of bilateral deafness appearing as a sequel to poliomyelitis.

Ayer and Trevett (1934) observed a case with a persistent

SUMMARY AND CONCLUSIONS

EPIDEMIOLOGY

(1) *Area* There is no evidence that the towns have a greater liability than the rural districts.

(2) *Season.* In this series the majority of cases occurred during July, August and September, and the duration of time between the onset of illness and the first appearance of the paralysis was about one week.

(3) *Contacts.* Thirty-five per cent of our cases gave a history of being in contact with another case of poliomyelitis and 30% had been in contact with minor respiratory illnesses.

(4) *Age.* Poliomyelitis in infants under 6 months of age is infrequent. Approximately two-thirds of this series were under 15 years and approximately one-third under 5 years. It was also apparent that adults were more severely affected than children.

(5) *Sex.* There was a slight preponderance of males over females.

(6) *Inoculations.* There is no doubt that there is an increased risk of a child developing a paralytic attack of poliomyelitis following intramuscular immunizing injections at a time when there is an epidemic of poliomyelitis. Therefore during the danger period, it is advisable to suspend temporarily prophylactic immunizing procedures.

Immunization against diphtheria should, however, preferably be carried out between the ages of 2 to 5 months.

(7) *Tonsillectomy.* My own observations would appear to support the view that bulbar lesions are more frequent in patients in whom poliomyelitis follows a recent tonsil-

- REDESCHI, E (1903) *Atti Accad. Sci. med. nat. Ferrara*, 78, 57.
RIDLON, J (1898). *Chicago med. Rec.*, 14, 436.
RUMSHINA, E (1933) *J. Bone Jt. Surg.*, 15, 772.
SCHIMOUCLER, G (1913) Paris thesis, 41, 54.
TILNEY, F. (1918). *Neurol. Bull.*, 1, 7.
TUMPEER, I. H., and MCNEALY, E. W. (1930). *J. Amer. med. Ass.*, 95, 19.
VALENTIN, B (1935) *Med. Welt.*, 7, 1061.
WAPPLER, R (1937) *Z. Orthop.*, 66, 43.

Swimming and Exercise The avoidance of strenuous exercise is imperative. Rest should be insisted upon during the prodromal symptoms

Operations and Inoculations These should be strictly avoided during an epidemic.

Deformities. Early treatment decreases the necessity for operation and also the incidence of scoliosis.

CLINICAL FEATURES

It must be stated definitely that laboratory investigations at present cannot replace careful clinical observation. This especially applies when one has to decide when a mechanical respirator should be used.

Signs and symptoms vary greatly in different epidemics. There is also a marked difference in the distribution and severity of the paralysis. The intensity and extent of muscular pain varies also from epidemic to epidemic and from one area to another. The pain may be relieved in one case and be quite unaffected by the same drug in another case.

If the patient is treated in hospital during the acute stage, progress is more rapid and the end results improved

ABORTIVE PHASE

The percentage of cases found in a series of five hundred which suffered from fever and rigors is given.

Epistaxis occurred in only one case, but a sore throat was complained of in 29% of cases. Catarrh of the nose and chest was present in 19% and 16% of cases respectively

PREPARALYTIC PHASE

Tachycardia was sometimes accompanied by a rise in the blood pressure and cardiac murmurs

Hypertension may be due to the destruction of the vasodilator centre in the medulla as found at post-mortem
2% of cases were comatose and 3% had generalized

lectomy. It is advisable to postpone tonsillectomy until the risk of an autumn recrudescence of the outbreak has passed.

(8) *Exercise.* From my own observations there is little doubt that physical activity after the onset of the preparalytic stage is highly dangerous. It is therefore imperative that during an epidemic of poliomyelitis minor illnesses should be treated by rest in bed and sedatives in the form of phenobarbitone gr. $\frac{1}{2}$ t.d.s. in an attempt to reduce restlessness. If the patient complains of pain in the head, limbs or spine, analgesics should be given. It would, however, appear that mental activity has little if any effect upon any spontaneous movement it may produce.

An abortive attack may be converted into a paralytic one when a symptomless patient is permitted severe physical activity. It would therefore seem reasonable to advise compulsory periods of rest during an epidemic.

PATHOLOGY

From the study of over five hundred cases of poliomyelitis it has become obvious that any part of the central nervous system, including the sympathetic and parasympathetic, can be involved. There is also little doubt that second attacks of poliomyelitis although rare can occur.

Macroscopic and microscopic examinations agree in general with those described by most competent observers, and it has been seen that the microscopic changes are more widespread than the clinical features.

Multiple cases of poliomyelitis in one family are fairly common, but one is usually of the paralytic type and the remainder of the abortive type.

PROPHYLAXIS

Crowds and Schools. During an epidemic, crowded meetings, parties, etc., should be avoided by everyone. The closure of schools, however, appears to be contra-indicated in the early stages, because children are then free to gather together and to play vigorous games.

Swimming and Exercise. The avoidance of strenuous exercise is imperative. Rest should be insisted upon during the prodromal symptoms.

Operations and Inoculations. These should be strictly avoided during an epidemic

Deformities Early treatment decreases the necessity for operation and also the incidence of scoliosis.

CLINICAL FEATURES

It must be stated definitely that laboratory investigations at present cannot replace careful clinical observation. This especially applies when one has to decide when a mechanical respirator should be used.

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PREPARALYTIC PHASE

Tachycardia was sometimes accompanied by a rise in the blood pressure and cardiac murmurs.

Hypertension may be due to the destruction of the vasodilator centre in the medulla as found at post-mortem. 2% of cases were comatose and 3% had generalized

convulsions; 41% suffered from insomnia and 5% were stuporose.

The eye signs varied greatly but in general were those described in the text. Papilloedema was present in a large number of our cases which were not of the bulbar type.

Gastro-intestinal atony was common, and whilst 56% had constipation 10% had diarrhoea.

As regards the bladder, 6% suffered from incontinence of urine, and 8% had retention. It was confirmed that retention of urine in poliomyelitis is relatively unknown in infants and children.

PARALYTIC PHASE

The number of days between the onset of the disease and the first appearance of paralysis is recorded.

In 50% the onset of paralysis was sudden and in 50% it was gradual.

In 55% of cases the paralysis increased during the first twenty-four hours, in 31% it was stationary, and in 14% it decreased in extent.

In 45% it was ascending in form, and in 55% it was descending.

In a few cases there was a temporary loss of sensation.

As regards the tendon reflexes, it was noted that a loss of the knee jerks may not be accompanied by any detectable paralysis.

(1) *Polioencephalitis*. The eye signs noted were as follows.

Unequal pupils	2%	4% nystagmus
Diplopia	6%	19% photophobia

Fifteen cases were found to have facial paralysis, one of which was bilateral.

Atypical electrocardiographic changes were also common in the bulbar form.

(2) *Cerebellar Form*. Acute cerebellar ataxia with no paralysis can occur. In this series, vertigo occurred in 18% of cases.

cerebrospinal fluid cell count with a preponderance of polymorphonuclears in the first few days followed later by a lymphocytosis and a diminished polymorphonuclear count by the end of the second week.

Against a diagnosis of poliomyelitis we noted:

- (1) Persistent absence of changes in the cerebrospinal fluid.
- (2) Persistent absence of pleocytosis or a pleocytosis over 1,000/cm.
- (3) Values of albumen over 200 mgm. %.
- (4) Decreased quantity of sugar, especially a progressive decrease of sugar during the illness.

BLOOD GROUPS

We failed to find that any particular blood group was unduly susceptible to infection by poliomyelitis.

MUSCLE

In our series it was soon obvious that the Medical Research Council's grading of muscle power was simple, required no apparatus and was remarkably accurate.

For the examination of muscle power in any one muscle or muscle group, we advise that it should not take more than two to three minutes, and that it should not be repeated more than twice in twenty-four hours.

In young children we encountered considerable difficulty in muscle testing cases in which muscular contractions were already present.

Jerky movements of the limbs were noted in 22% of our cases and twitching of individual muscles in 12%.

Pain in the limbs occurred in 18% of cases.

Pain and tenderness on stretching a muscle group was not a common finding and is usually observed after the paralysis is well established.

Muscle tenderness lasted in the majority of our cases for periods of from one day to one week whilst the minority lasted up to five weeks.

Muscle tenderness was particularly noted in sixteen cases.

Thus:

- 5 cases had slight tenderness with severe subsequent paralysis.
- 6 cases had fairly severe tenderness with no subsequent paralysis.
- 3 cases had fairly severe tenderness with slight subsequent paralysis.
- 2 cases had slight tenderness with slight subsequent paralysis.

In our opinion, there are two types of muscle spasm:

- (1) Early spasmodic which occurs in the acute phase and lasts for a few days. In other words it is fairly transitory
- (2) This type persists for more than a month and is very troublesome. It appears to be a reflex contracture due to a pain stimulus, and is frequently accompanied by *stiff joints and contractures*.

The various theories of muscle spasm are discussed.

There appears to be no relationship between the degree of paralysis and the frequency and intensity of muscle spasm.

There is no definite proof that the relief of muscle spasm prevents paralysis or alters the mortality in the acute stage.

Spasm temporarily impairs muscle function and destroys the normal reciprocal relation between agonists and antagonists.

Marked rigidity was present in 22% of cases, Kernig's sign in 20% of cases, head retraction in 18% and stiff back in 62% of cases.

Muscle spasm was aggravated by massage, premature weight bearing and exposure to cold.

PARESIS AND PARALYSIS

Fifty per cent of our cases were of sudden onset and 50% were gradual.

Muscular weakness in poliomyelitis has been attributed to several causes some of which are enumerated in the text.

The muscles appear to be involved in proportion to their

activity or fatigue and the most highly developed muscles are those most severely affected.

An ascending type of paralysis was present in 30% to 45% of cases in all age groups and a descending type in 55%.

In 80% of cases, the paralysis progressed for twenty-four hours

In 80% of cases with paralysis of the abdominal muscles, this was bilateral

The return of muscle power has been attributed to several factors.

Advice is given on muscle testing and charting.

A complete muscle chart is made out on admission and at repeated regular intervals. The whole course of treatment is based on this chart.

Comprehensive notes are given on individual muscle paresis and paralysis

COMPLICATIONS

In the age group 0-5 years, by far the most common complication was genu valgum. Genu recurvatum occurred in 8 cases, and pes planus and equinus deformity of the foot in 4 cases. Scoliosis occurred in only 3 cases.

In the age group 5-15 years, the most common complication was scoliosis whilst deformities of the lower limb were relatively rare

In the over 15 age group, deformities of the spine were again the most common, but were only 50% greater than deformities of the lower limb.

DIFFERENTIAL DIAGNOSIS

An attempt is made to describe the differential diagnosis, and the leading signs and symptoms are given.

TREATMENT

The poliomyelitis team should wherever possible consist of a physician, paediatrician, neurologist, orthopaedic surgeon, physical medicine specialist, physiotherapist, occupational therapist and almoner. Every member of the team

contributes his or her special skill for the ultimate benefit of the patient

Each case must be dealt with as an individual problem in order to obtain the best results.

A résumé is given of the essential points of treatment.

There appears to be little doubt that the earlier the diagnosis is made, and complete rest ordered, then the less permanent damage to the neuromuscular units will occur.

Deformities can be prevented by properly supervised orthopaedic treatment which must, however, be started before contractures can develop. The treatment must be supervised over a number of years, so preventing the onset of late deformities. Short periods of rehabilitation can be given at yearly intervals if required.

There is no doubt that inefficient treatment in the early stages greatly lengthens the patient's stay in hospital

Muscle imbalance and deformities are common in paralytic poliomyelitis and frequently mechanical aids or operation are required

TRACHEOTOMY

The indications for tracheotomy are given

In our experience, if tracheotomy is performed early, it is more effective than postural drainage and aspiration alone

An Ear, Nose and Throat specialist is considered ideal for the successful treatment of respiratory complications. Once complications have developed, the value of tracheotomy decreases rapidly if cyanosis has occurred or if a respirator is required.

Tracheotomy relieves respiratory obstruction caused by

iii immobilization is explained in detail.

DRUGS AND SERUM

The appropriate drugs and serum used in poliomyelitis are listed but none are found to be specific

NURSING

Details are given of the appropriate nursing treatment including the use of a mechanical respirator.

MUSCLE RE-EDUCATION

A brief description is given of muscle re-education. Its aim is to produce hypertrophy of the remaining muscle fibres.

Controlled muscle activity and exercise to the unaffected part of the body are given to maintain tone and to prevent disuse atrophy.

Details of thermotherapy and pool therapy are given.

Passive, active and resisted movements are mentioned.

Trick movements and fatigue are most important and are particularly mentioned

The part played by electrotherapy and massage is assessed.

For completeness, the Sister Kenny treatment is discussed, but we do not favour it and it has not been used in any of our cases.

Treatment in the intermediate stage is given in fair detail.

The part played by class exercises and games and occupational therapy is also given.

Mention is made of the Residual stage, but as the treatment is usually surgical, details are not given.

PREGNANCY

Pregnancy has no influence on the severity of the disease or on the residual paralysis, and does not increase the risk of death.

About 50% of maternal deaths occur during the last trimester and in the immediate puerperium.

The degree of spinal paralysis has no serious effect on labour, and the lack of voluntary muscular effort does not contra-indicate vaginal delivery.

There is no reason to terminate pregnancy before term, except on purely obstetrical grounds. Pregnancy, labour and delivery are conducted along orthodox obstetrical lines. If the patient should become markedly distressed, the respira-

tory embarrassment will be due solely to the disease and not to the accompanying pregnancy.

It would appear that Caesarean section is used more often than ■ necessary. It is rarely indicated, and then only when some obstetrical complication occurs, or to obtain a viable child when the mother's life will obviously be lost.

In my experience, poliomyelitis and pregnancy together are more frequently found in the last two trimesters.

The combination of poliomyelitis and pregnancy, unlike that of rubella and pregnancy, does not appear to result in congenital abnormalities in the child

It would appear to be true that severely ill patients die from the disease itself and not because it is associated with pregnancy.

Death may be due to

- (1) Respiratory centre paralysis
- (2) Paralysis of the primary muscles of respiration.
- (3) Obstruction of the larynx due to.
 - (a) paralysis,
 - (b) aspiration of secretions,
 - (c) pulmonary oedema.
- (4) Pulmonary complications.
 - (a) atelectasis,
 - (b) bronchopneumonia,
 - (c) marked congestion of the lungs.

Examination of our figures showed that the most common cause of death was pulmonary oedema and hyperpyrexia

APPENDIX "A"

POLIOMYELITIS UNIT MUSCLE CHART

(1) NECK, SHOULDER GIRDLE AND ARM

NAME _____

RECORD No.

DATE OF BIRTH

WARD

DATE OF ONSET

DATE OF COMMENCEMENT OF TREATMENT IN UNIT

RIGHT SIDE					LEFT SIDE			
				Examiner				
				Dates				
				STERNOMASTOIDS				
				SCALENI				
				FLEXORS OF NECK				
				ERECTOR SPINAE—CERV.				
				TRAPEZIUS—UPPER FIBRES				
				TRAPEZIUS—LOWER FIBRES				
				SERRATUS ANTERIOR				
				LEVATOR SCAPULAE				
				RHOMBIOIDS				
				LATISSIMUS DORSI				
				PECTORALIS MAJOR AND MINOR				
				DELTOID—ANT FIBRES				
				DELTOID—MIDDLE FIBRES				
				DELTOID—POST FIBRES				
				SUPRASPINATUS				
				EXT ROTATORS SHOULDER				
				INT ROTATORS SHOULDER				
				BICEPS				
				BRACHIALIS				
				TRICEPS				

RANGE OF PASSIVE MOVEMENTS OF JOINTS

RIGHT SIDE						LEFT SIDE				
					Examiner					
					Dates					
					SHOULDER					
					FLEXION					
					EXTENSION					
					ABDUCTION					
					ADDUCTION					
					MEDIAL ROTATION					
					LATERAL ROTATION					
					ELBOW					
					FLEXION					
					EXTENSION					

REMARKS

STRENGTH OF CONTRACTION:

- 0 — NO CONTRACTION
- 1 — FLICKER
- 2 — CONTRACTION BUT NOT AGAINST GRAVITY
- 3 — CONTRACTION AGAINST GRAVITY ONLY
- 4 — CONTRACTION AGAINST GRAVITY AND RESISTANCE
- 5 — NORMAL CONTRACTION

APPENDIX "A"

POLIOMYELITIS UNIT MUSCLE CHART

(1) NECK, SHOULDER GIRDLE AND ARM

NAME

RECORD No.

DATE OF BIRTH

WARD

DATE OF ONSET

DATE OF COMMENCEMENT OF TREATMENT IN UNIT

RIGHT SIDE								LEFT SIDE			
				EXERCISE							
				Dorsi							
				STERNOMASTOIDS							
				SCALENI							
				FLEXORS OF NECK							
				ERECTOR SPINAE—CERV							
				TRAPEZIUS—UPPER FIBRES							
				TRAPEZIUS—LOWER FIBRES							
				SERRATUS ANTERIOR							
				LEVATOR SCAPULAE							
				RHOMBOIDS							
				LATISSIMUS DORSI							
				PECTORALIS MAJOR AND MINOR							
				DELTOID—ANT FIBRES							
				DELTOID—MIDDLE FIBRES							
				DELTOID—POST FIBRES							
				SUPRASPINATUS							
				EXT ROTATORS SHOULDER							
				INT ROTATORS SHOULDER							
				BICEPS							
				BRACHIALIS							
				TRICEPS							

RANGE OF PASSIVE MOVEMENTS OF JOINTS

RIGHT SIDE							LEFT SIDE					
						EXTENSION						
						DEGREE						
						FOREARM						
						PRONATION						
						SUPINATION						
						WRIST						
						FLEXION						
						DORSIFLEXION						
						ABDUCTION						
						ADDUCTION						

REMARKS

STRENGTH OF CONTRACTION:

- 0 — NO CONTRACTION
- 1 — FLICKER
- 2 — CONTRACTION BUT NOT AGAINST GRAVITY
- 3 — CONTRACTION AGAINST GRAVITY ONLY
- 4 — CONTRACTION AGAINST GRAVITY AND RESISTANCE
- 5 — NORMAL CONTRACTION

APPENDIX "B"

POLIOMYELITIS UNIT MUSCLE CHART

(2) FOREARM AND HAND

NAME

RECORD No.

DATE OF BIRTH

WARD

DATE OF ONSET

DATE OF COMMENCEMENT OF TREATMENT IN UNIT

RIGHT SIDE					LEFT SIDE			
				Examiner				
				Dates				
				BRACHIORADIALIS				
				SUPINATOR				
				PRONATOR TERES				
				FLEXOR CARPI RADIALIS				
				FLEXOR CARPI ULNARIS				
				PALMARIS LONGUS				
				EXT CARPI RAD LONGUS				
				EXT CARPI RAD BREVIS				
				EXT CARPI ULNARIS				
				FLEXOR DIGITORUM SUB				
				FLEXOR DIGIT PROF 1				
				" " " 2				
				" " " 3				
				" " " 4				
				FLEXOR POLLICIS LONGUS				
				FLEXOR POLLICIS BREVIS				
				ABDUCTOR POLLICIS LONGUS				
				ABDUCTOR POLLICIS BREVIS				
				ADDUCTOR POLLICIS				
				OPPONENS POLLICIS				
				ABD MINIMI DIGITI				
				OPPONENS MINIMI DIGITI				
				EXT DIG COMMUNIS				
				EXTENSOR INDICIS				
				EXT MINIMI DIGITI				
				EXT POLLICIS LONGUS				
				EXT POLLICIS BREVIS				
				DORSAL INTEROSSEI				
				PALMAR INTEROSSEI				
				LUMBRICALES				

RANGE OF PASSIVE MOVEMENTS OF JOINTS

RIGHT SIDE						LEFT SIDE				
					EXAMINER					
					Dates					
					HIP					
					FLEXION					
					EXTENSION					
					ABDUCTION					
					ADDUCTION					
					MEDIAL ROTATION					
					LATERAL ROTATION					
					KNEE					
					FLEXION					
					EXTENSION					
					ANKLE					
					DORSIFLEXION					
					PLANTARFLEXION					
					INVERSION OF FOOT					
					EVERSION OF FOOT					

REMARKS

STRENGTH OF CONTRACTION*

- 0 — NO CONTRACTION
- 1 — FLICKER
- 2 — CONTRACTION BUT NOT AGAINST GRAVITY
- 3 — CONTRACTION AGAINST GRAVITY ONLY
- 4 — CONTRACTION AGAINST GRAVITY AND RESISTANCE
- 5 — NORMAL CONTRACTION

APPENDIX "C"

POLIOMYELITIS UNIT MUSCLE CHART

(3) TRUNK AND LEG

NAME _____

RECORD NO.

DATE OF BIRTH

WARD

DATE OF ONSET

DATE OF COMMENCEMENT OF TREATMENT IN UNIT

[illegible]

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APPENDIX "D"

OTHER important points which may be incorporated either in the muscle chart or on the patient's case sheet are:

- (1) Cranial nerve involvement.—All that is necessary is to denote which nerves are involved, because it is impossible to test the strength of contraction of the ocular muscles, tongue muscles, etc.
- (2) Presence and site of muscle tenderness and pain, and whether the pain is worse when the muscle is stretched.
- (3) Degree of limitation of active movement of any particular joint.
- (4) Presence of contractures and deformities.
- (5) Dates when patient assumes sitting and standing posture.
- (6) Dates when patient assumes walking with appliances and crutches.
- (7) Dates when patient discards appliances and/or crutches
- (8) Type of gait.
- (9) Chest expansion at nipple line, and the vital capacity. This is an indication of the involvement of the diaphragm and the intercostals and also of their recovery.
- (10) The following measurements are also important:
 - (a) Length of legs from anterior superior spine to medial malleolus.
 - (b) Circumference of thigh 6 inches above the patella.
 - (c) Circumference of calf 6 inches below patella
- (11) Date and nature of operations.

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